EFFECTS OF CHEST WALL CONSTRICTION ON AEROBIC CAPACITY DURING EXERCISE

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I have examined the final copy of this Thesis for form and content and recommend that it be accepted in partial fulfillment of the requirement for the degree of Master of Education, with a major in Physical Education-Exercise Science.

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We have read this Thesis
and recommend its acceptance

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DEDICATION

This thesis is dedicated to my parents in appreciation for all the love and support they have given me throughout the years.

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ABSTRACT

The aim of the present study was to determine whether a reduction in lung volume and chest wall movement using an externally applied thoracic constriction band over an eight-week training period, could improve aerobic capacity and running performance. Participants included 22 healthy, non-active adults (26 ± 4 year (mean ± SD)) who were studied over a period of eight weeks during which they participated in aerobic exercise for three days/week for 30 minutes/session at a moderate intensity estimated at 65% to 80% of their maximal exertion. Aerobic capacity (VO_{2max}) and pulmonary lung function (FVC, FEV_1, $FEF_{25-75\%}$, PEF) were measured pre-, mid-, and post- eight weeks. Following the pre-testing, participants were randomly placed into one of two groups: (1) Chest Wall Restriction (CWR), or (2) a control group (Non-Chest Wall Restriction (NCWR)). The CWR Group performed the exercise sessions while wearing an elastic strap. Participants were encouraged to use a treadmill, elliptical machine, and/or stationary cycle in random order to complete the exercise session. Pulmonary lung function measures were not changed over the eight weeks for either group. At eight weeks, CWR and NCWR groups had significant increases in VO_{2max} (from 33.55 ± 6.48 to 37.78 ± 7.11 and 33.30 ± 10.39 to 35.99 ± 9.09 $ml \cdot min^{-1} \cdot kg^{-1}$ ± SD, respectively). However, a significant improvement of 11.0 ± 4.0% in aerobic capacity was observed in the experimental group at just four weeks compared to the control group (3.0 ± 6.0%, $P < 0.05$). These results suggest that the use of an externally applied thoracic constriction band during aerobic exercise assists in increasing aerobic capacity more rapidly than training without a band, because of the greater increase in VO_{2max} seen within the first four weeks for the CWR group compared to the NCWR group.

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LIST OF ABBREVIATIONS

atm	Atmosphere
bpm	Breathes per Minute
CO_2	Carbon Dioxide
COPD	Chronic obstructive pulmonary disease
CWR	Chest Wall Restriction Group
ERV	Expiratory reserve volume
$FEF_{25-75\%}$	Forced expiratory flow from 25% to 75% of the vital capacity
FEV_1	Forced expiratory volume in one second
FVC	Forced vital capacity
FRC	Functional residual capacity
IMT	Inspiratory muscle training
NCWR	Non-Chest Wall Restriction Group
NMDs	Neuromuscular Disorders
mm Hg	Millimeters of Mercury
MVV	Maximal voluntary ventilation
PEF	Peak expiratory flow rate
P_{CO2}	Partial Pressure of Carbon Dioxide
Pdi	Transdiaphrgmatic Pressure
PE_{max}	Maximal Expiratory Mouth Pressure
PI_{max}	Maximal Inspiratory Mouth Pressure
P_m	Peak pressure
P_{O2}	Partial Pressure of Oxygen

RV	Residual volume
TLC	Total lung capacity
VC	Vital capacity
VO_{2max}	Maximal Volume of Oxygen
V_T	Tidal volume

CHAPTER 1

INTRODUCTION

Any skeletal muscle in the human body can be made to fatigue, including the muscles used to assist in breathing such as the diaphragm and accessory ventilatory muscles (Bellemare and Grassino, 1982a; Bellemare and Grassino, 1982b; Cohen et al., 1982). In a laboratory setting with spirometry equipment, one could show respiratory muscle fatigue after two minutes of maximum voluntary ventilation (MVV) breathing (Rafferty et al., 1999). Maximum voluntary ventilation tests repeated in succession also usually produce decreasing values.

Individuals with respiratory disease, such as chronic obstructive pulmonary disease or other disorders that may involve the chest wall, such as ankylosing spondylitis, have often been the populations studied when attempting to answer questions regarding the relationship between fatigue in the ventilatory muscles and the performing body. Chest constriction (external thoracic restriction) studies have primarily been conducted to assess the factors that may contribute to the dyspnea (shortness of breath) observed in clinical conditions that result in restrictive lung and/or chest wall changes and a reduced intrathoracic space (Harty et al., 1999; O'Donnell et al., 2000).

Special breathing exercises, such as pursed-lip breathing, help mitigate dyspnea (Opdekamp and Sergysels, 2003). Studies have shown that the use of breathing exercises results in a slower, deeper ventilatory pattern that improves oxygenation (Dechman and Wilson, 2004) by improving ventilation-perfusion matching within the lungs (Donado and Hill, 1988). Breathing against a flow or threshold resistor or doing maximum voluntary ventilation maneuvers several times a day has also been shown to strengthen

the ventilatory muscles in clinical populations (McConnell and Romer, 2004). However, the use of chest constriction as a mode of muscular conditioning to enhance performance in healthy adults has not been studied. It is unlikely that resistive inspiration exercises alone can improve exercise tolerance in healthy adults, but they may be able to assist in a more rapid adaptation to physical exertion coming from a sedentary lifestyle.

1.1 Statement of the Problem

Studies have indicated that the respiratory skeletal muscles, like muscles of the limbs, fatigue under conditions of intense activity, leading to respiratory failure (Bellemare and Grassino, 1982a; Bellemare and Grassino, 1982b; Cohen et al., 1982). Special breathing exercises, breathing against a flow or threshold resistor, or doing maximum voluntary ventilation maneuvers several times a day has been shown to strengthen the ventilatory muscles in the clinical populations (McConnell and Romer, 2004). The use of chest constriction has been shown to limit exercise performance (Vanmeenen et al., 1984; Hussain and Pardy, 1985; Hussain et al., 1985; O'Donnell et al., 2000; Coast and Cline, 2004; Wang and Cerny) and pulmonary function (Hussain and Pardy, 1985; Hussain et al., 1985; Harty et al., 1999; O'Connor et al., 2000; O'Donnell et al., 2000; Wang and Cerny, 2004), but it has not been studied as a mode of muscular conditioning to enhance performance in healthy adults.

1.2 Purpose

The purpose of this study was to determine whether a using an externally applied thoracic constriction band to reduce lung volume and chest wall movement over an eight-week training period could improve aerobic capacity and running performance.

1.3 Significance of the Study

Studies have shown that chest restriction (external thoracic restriction) during moderate intensity exercise limits an individual's respiratory muscle function (Hussain and Pardy, 1985; Hussain et al., 1985; Harty et al., 1999; O'Connor et al., 2000; O'Donnell et al., 2000; Wang and Cerny, 2004) and exercise performance (Vanmeenen et al., 1984; Hussain and Pardy, 1985; Hussain et al., 1985; O'Donnell et al., 2000; Coast and Cline, 2004; Wang and Cerny). However, available information regarding the use of chest restriction as a training mode during exercise has yet to be tested. The findings from this study will provide information as to whether the use of chest restriction during exercise may enhance or hinder aerobic capacity and performance. If shown to improve these components, the use of chest restriction could be introduced into the non-active population as a mode of training.

1.4 Variables

1.4.1 Independent Variable

The independent variable of this study was the externally applied thoracic constriction band worn around the chest of 22 healthy, non-active adults during aerobic exercise three days per week for eight weeks. A control group that did not wear the constriction band was monitored in the same training for the same period of time.

1.4.2 Dependent Variable

The dependent variables for this study were pulmonary function, forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), forced expiratory flow from 25 to 75 percent of the vital capacity ($FEF_{25-75\%}$), peak expiratory flow (PEF), VO_{2max}, and time to exhaustion.

1.4.3 Other Variables

The control variables for this study were age, gender, and history of exercise. The extraneous variables for this study were participants' other daily physical activities.

1.5 Research Hypothesis

It was hypothesized that participation in aerobic exercise for eight weeks while wearing an externally applied thoracic constriction band around the chest would result in improvements in aerobic capacity.

1.6 Assumptions

It was assumed that all individuals volunteering to participate in the study would offer their best effort during pretest, mid-test, and posttest assessments. The research technician offered verbal encouragement to participants throughout testing. To ensure maximum effort and complete understanding of the protocol, a research technician thoroughly explained to each participant what was expected. Each participant completed an exercise log sheet after each session stating his/her activities. A research technician placed individual telephone calls or sent e-mails weekly to encourage compliance and answer questions.

1.7 Limitations

Results of this study may have been affected by the compliance of the participants that volunteered. Some participants may have participated in additional exercise activities during personal time throughout the eight-week study. Participants may have become unmotivated and stopped participating in the testing if the program became boring or challenging. Additionally, those participants who for various reasons exited the study prior to post testing, may have limited conclusions based on the final data.

1.8 Delimitations

The results of this study are limited to healthy, non-active adults 18 to 35 years of age and may not be fundamental to other age groups. Results pertain to the aerobic training protocol and testing equipment used in this study. Therefore, other protocols using the same methods and/or different testing equipment may not deliver the same results. Volunteers used in this study were non-active individuals and were not necessarily familiar with motivating themselves beyond physical and psychological discomfort. Therefore, participants' may not have reached true maximums during pre/mid/post testing and desired exercise protocol intensity may not have been achieved.

1.9 Definitions

1. Aerobic exercise: The ability of the heart, lungs, and blood vessels to deliver an adequate supply of oxygen while exercising large muscle mass, performing rhythmic activity, walking, jogging, biking, swimming, etc. (Cotton, 1996).

2. Expiratory reserve volume (ERV): The maximum volume of air that can be exhaled from the end-expiratory level or from functional residual capacity (FRC) to residual volume (Reid and Chung, 2004).

3. Forced expiratory flow from 25% to 75% of the vital capacity ($FEF_{25-75\%}$): The average flow rate during the middle half of the forced expiratory vital capacity (Reid and Chung, 2004).

4. Forced expiratory volume in one second (FEV_1): The maximum volume of air that can be expired from a full inspiration in one second (Miller, 1987).

5. Forced vital capacity (FVC): The total volume of air exhaled with a maximal forced expiratory effort after a full inspiration (Reid and Chung, 2004).

6. Functional residual capacity (FRC): The volume of air remaining in the lungs at the end of an ordinary expiration (Reid and Chung, 2004).

7. Inspiratory muscle training (IMT): Used to improve strength and endurance of the inspiratory muscles (Covey et al., 2001).

8. Maximal voluntary ventilation (MVV): The volume of air that a subject can breath upon repetitive maximal voluntary effort (Miller, 1987).

9. Peak expiratory flow rate (PEF): The highest flow rate obtained during a forced expiratory maneuver (Reid and Chung, 2004).

10. Peak pressure (P_m): (also known as: inspiratory muscle endurance) the pressure achieved with the heaviest load until the subject is exhausted and can no longer inspire (Inbar et al., 2000).

11. Residual volume (RV): The volume of air remaining in the lungs after a maximal expiration (Reid and Chung, 2004).

12. Respiratory muscle fatigue: Loss in the capacity to develop force and/or velocity of a muscle, resulting from muscle activity under load and is reversible by rest (NHLBI, 1990).

13. Respiratory muscle weakness: Impairment in the capacity of a fully rested muscle to generate force (NHLBI, 1990).

14. Tidal volume (V_T): The volume of air inhaled and exhaled during breathing (Reid and Chung, 2004).

15. Total lung capacity (TLC): The total amount of air in the lungs after a maximal inspiration (Reid and Chung, 2004).

16. Vital capacity (VC): The maximum volume of air that can be expelled after a maximum inspiration (Reid and Chung, 2004).

17. VO_{2max}: The maximum rate of oxygen utilization of muscles during exercise (Heyward, 2002).

CHAPTER 2

LITERATURE REVIEW

2.1 Physiology of the Respiratory System

The coordinated action of a relatively large number of muscles produces breathing (Cherniack and Widdicombe, 1986). The respiratory system consists of two portions: (1) the upper respiratory system, including the nose, pharynx, and associated structures and (2) the lower respiratory system, including the larynx, trachea, bronchi, and lungs (see Figure 2.1). The major function of the respiratory system is to supply the body with oxygen (O_2) and dispose of carbon dioxide (CO_2). To accomplish this, respiration, which consists of these processes, must occur (Marieb, 2004).

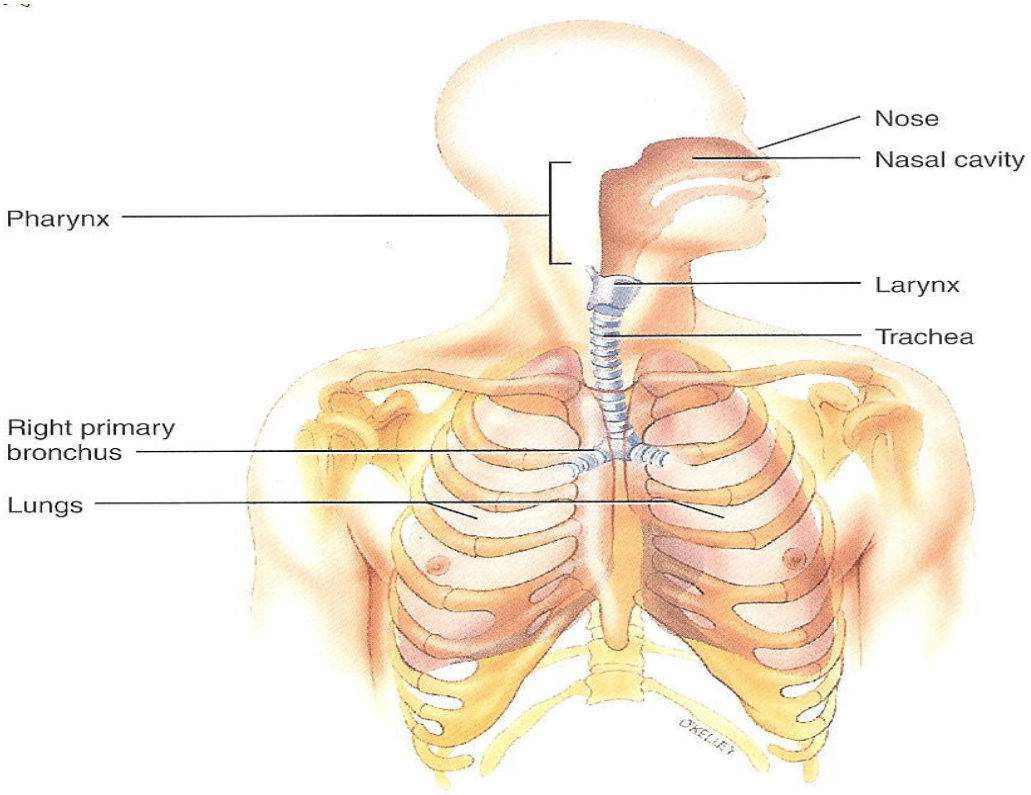

Figure 2.1. Anterior view showing organs of respiration (Tortora and Grabowski, 2000).

2.2 Respiration

Respiration is the process of gas exchange in the body. This is composed of three basic stages: (1) pulmonary ventilation, (2) external respiration, and (3) internal respiration (Tortora and Grabowski, 2000).

2.2.1 Pulmonary Ventilation

Pulmonary ventilation is the mechanical flow of air into (inspiration) and out of (expiration) the lungs, which is commonly known as breathing. In this process, gases are exchanged between the atmosphere and lung alveoli. Air flows between the atmosphere and the lungs because of alternating pressure differences created by the contraction and relaxation of the respiratory muscles (Tortora and Grabowski, 2000). The rate of airflow and the amount of effort needed for breathing is also influenced by alveolar surface tension, compliance of the lungs, and airway resistance.

2.2.1.1 Inspiration

Prior to inspiration, air pressure within the lungs is equal to the pressure of the atmosphere (sea level is 760 millimeters of mercury (mm Hg), or 1 atmosphere (atm)). For air to flow into the lungs, the pressure inside the alveoli must be lower than the atmospheric pressure (Tortora and Grabowski, 2000). This is achieved by increasing the volume of the lungs.

Differences in pressure, caused by changes in lung volume, force air into the lungs during inhalation and out during exhalation. For inspiration to occur, the lungs must expand, increasing their volume and decreasing the pressure in the lungs to below atmospheric pressure (Tortora and Grabowski, 2000). The relationship between the pressure and volume of gas is defined by Boyle's law (Marieb, 2004). Boyle's Law

indicates that for a fixed amount of gas (fixed number of moles) at a fixed temperature, the pressure and the volume are inversely proportional. In other words, as the pressure increases, the volume decreases (i.e., when a balloon is squeezed to increase the pressure, the volume of the balloon goes down.).

During normal breathing, the pressure between the two pleural layers, called intraplueral pressure, is always subatmospheric (i.e., lower than atmospheric pressure). Just before inspiration, intraplueral pressure is approximately 756 mm Hg if the atmospheric pressure is 760 mm Hg (Tortora and Grabowski, 2000). As the diaphragm contracts and the thoracic cavity begins to increase in size, intraplueral pressure decreases to approximately 754 mm Hg (Tortora and Grabowski, 2000). As the volume of the lungs increases, pressure within the lungs (alveolar pressure) decreases from 760 mm Hg to 758 mm Hg, causing a pressure difference between the atmosphere and the alveoli (Tortora and Grabowski, 2000). Inspiration then occurs as air flows from a region of higher pressure to a region of lower pressure, and air continues to flow into the lungs as long as this pressure difference exists (see Figure 2.2).

Expansion of the lungs involves multiple stages. The diaphragm and inspiratory muscles first contract. The diaphragm muscle is a dome-shaped skeletal muscle that separates the thoracic cavity from the abdominal pelvic cavity and forms the floor of the thoracic cavity. The diaphragm muscle originates form the xiphoid process and the anterior surfaces of the lumbar vertebrae. Contraction of the diaphragm causes it to flatten, compressing the abdominalpelvic cavity and expanding the thoracic cavities. Inspiratory muscles increase the size of the thoracic cavity by contraction of the external intercostal muscles that lifts and pulls the sternum superiorly (Marieb, 2004).

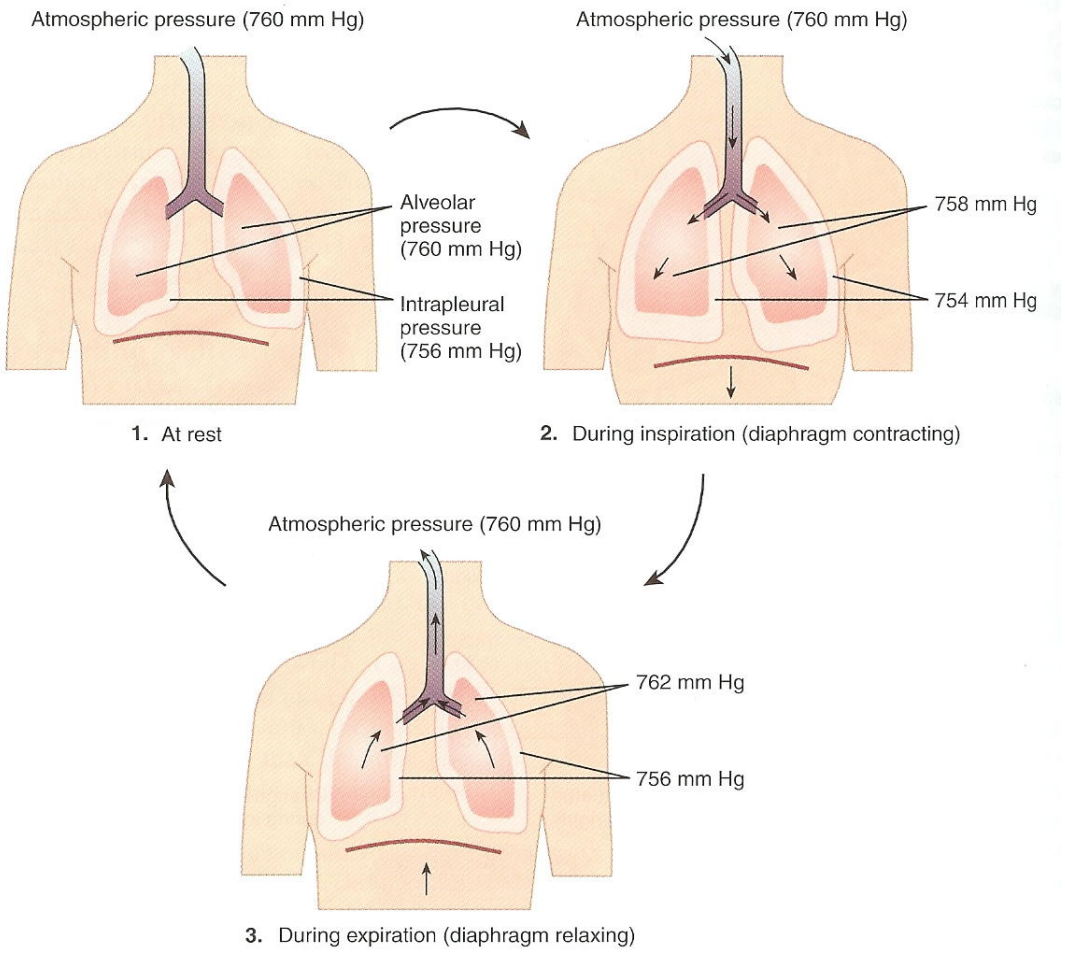

Figure 2.2. Pressure changes in ventilation (Tortora and Grabowski, 2000).

2.2.1.2 Expiration

Expiration is also due to a pressure gradient, but the gradient is reversed. The pressure in the lungs is greater than the pressure of the atmosphere. Unlike inspiration, no muscular contractions are involved at rest. Expiration results from elastic recoil of the chest and lungs, both of which have a natural tendency to return to the resting state.

As inspiration stops, expiration begins when the inspiratory muscles relax. As the diaphragm relaxes, its elastic properties cause its dome to push superiorly (Tortora and Grabowski, 2000). This movement decreases the vertical and anterior-posterior dimensions of the thoracic cavity, thus decreasing lung volume. Sequentially, the alveolar

pressure increases to approximately 762 mm Hg and air then flows from the higher region of pressure in the alveoli to the of lower pressure in the atmosphere around the individual (Tortora and Grabowski, 2000) (see Figure 2.2).

2.2.2 External Respiration

External respiration is the exchange of oxygen (O_2) and carbon dioxide (CO_2) between air in the alveoli of the lungs and blood in the pulmonary capillaries. Dalton's law states that each gas in a mixture of gases exerts its own pressure as if all the other gases were not present. The pressure of a specific gas in a mixture is called its partial pressure (P_x) (Tortora and Grabowski, 2000). The partial pressure of oxygen (P_{o2}) in alveolar air is 105 mm Hg, and at rest the P_{o2} of deoxygenated blood entering the pulmonary capillaries is approximately 40 mm Hg (Tortora and Grabowski, 2000).

External respiration in the lungs results in the conversion of deoxygenated blood to oxygenated blood. During inspiration, atmospheric air containing O_2 enters the alveoli. Deoxygenated blood is then pumped through the pulmonary arteries into the pulmonary capillaries surrounding the alveoli. The partial pressures of gases in the pulmonary capillaries then become equal to the partial pressures of gases in the alveolar air thus resulting in oxygenated blood.

During expiration, CO_2 is exhaled into the atmosphere. While O_2 is being diffused from the alveoli into deoxygenated blood, CO_2 is diffusing from the deoxygenated blood to the alveoli. The partial pressure of carbon dioxide (P_{CO2}) of deoxygenated blood is 45 mm Hg, whereas the P_{CO2} of alveolar air is 40 mm Hg (Tortora and Grabowski, 2000). Due to this difference in P_{CO2}, carbon dioxide diffuses from the deoxygenated blood into

the alveoli until the P_{CO2} of the blood decreases to 40 mm Hg (Tortora and Grabowski, 2000).

2.2.3 Internal Respiration

Internal respiration is the exchange of O_2 and CO_2 between the systemic capillaries and tissue cells. During this stage, oxygenated blood is converted into deoxygenated blood. Oxygenated blood entering the tissue capillaries has a P_{O2} of 100 mm Hg, whereas the tissue cells have an average P_{O2} of 40 mm Hg (Tortora and Grabowski, 2000). Because of the P_{O2} difference, oxygen diffuses from the oxygenated blood through the interstitial fluid and into tissue cells until the P_{O2} in the blood is reduced to 40 mm Hg.

While O_2 diffuses from the tissue capillaries into the tissue cells, CO_2 is diffusing from the tissue cells to the tissue capillaries. Tissue cells have an average P_{CO2} of 45 mm Hg, and the P_{CO2} of tissue capillary oxygenated blood is 40 mm Hg (Tortora and Grabowski, 2000). As a result, CO_2 diffuses from the tissue cells through interstitial fluid until the P_{CO2} in the blood increases to 45 mm Hg, thus deoxygenating the blood (Tortora and Grabowski, 2000). The deoxygenated blood returns to the heart where it is pumped to the lungs for another cycle of external respiration.

2.3 Factors Affecting Pulmonary Ventilation

Three factors affect the rate of airflow and the ease of pulmonary ventilation: (1) surface tension of alveolar fluid, (2) compliance of the lungs, and (3) airway resistance.

2.3.1 Surface Tension of Alveolar Fluid

A thin layer of alveolar fluid coats the luminal surface of alveoli and exerts a force referred to as surface tension. Surface tension occurs at any gas-liquid boundary

because the molecules of the liquid have a stronger affinity to each other than to gas molecules in the air (Marieb, 2004). When liquid surrounds the alveolus, surface tension produces an inwardly directed force causing the alveoli to assume the smallest diameter possible (Tortora and Grabowski, 2000). During inspiration, surface tension must be overcome to expand the lungs. Surface tension accounts for two-thirds of lung elastic recoil (Tortora and Grabowski, 2000). As detailed above, expiration reduces the volume of the thoracic cavity, which decreases the size of the alveoli.

2.3.2 Compliance of the Lungs

Compliance refers to the effort required to stretch the lungs and thoracic wall (Tortora and Grabowski, 2000). In the lungs, compliance is related to two factors: elasticity and surface tension. High compliance means that the lungs and thoracic wall expand easily, because the elastic fibers in lung tissue are easily stretched and surfactant in alveolar fluid reduces surface tension. Surfactant is a detergent-like complex of lipids and proteins produced by type II alveolar cells. Surfactant decreases the cohesiveness of water molecules (Marieb, 2004). Low compliance means that the lungs resist expansion, which is caused by certain pulmonary conditions that do the following: (1) scar lung tissue, (2) cause lung tissue to become filled with fluid, (3) causes deficiency in surfactant, or (4) impede lung expansion in any way.

2.3.3 Airway Resistance

Airflow through airways depends on both pressure difference and resistance (Tortora and Grabowski, 2000). Airflow resistance is calculated (2.0) as

$$Raw = P_A - Pao/V \qquad (2.0)$$

where V equals airflow (L/s), P_A equals alveolar pressure (cm H_2O), Pao equals airway-opening pressure (cm H_2O), and Raw equals airway resistance (cm H_2O/L/s) (Fishman et al., 1998). Walls of the airway, especially the bronchioles, offer some resistance to the normal flow of air into and out of the lungs. Resistance is decreased as the lungs expand during inhalation and the bronchioles dialate (Tortora and Grabowski, 2000). Airway resistance then increases during exhalation as the diameter of the bronchioles decrease. In addition, the degree of contraction and relaxation of smooth muscle that surrounds the airways regulates airway diameter and thus resistance. Signals from the sympathetic division of the autonomic nervous system trigger contraction of the smooth muscle (narrowing the airway and increasing resistance) or relaxation (dialating the airway and decreasing resistance) (Tortora and Grabowski, 2000).

Any condition that narrows or obstructs the airway increases resistance; thus more pressure is then required to maintain the same airflow. This is common in diseases such as asthma or chronic obstructive pulmonary diseases, because of an increased airway resistance due to obstruction or collapsed airways.

2.4 Pulmonary Diseases

2.4.1 Obstructive Diseases

Airway obstruction is an increased resistance to airflow and can be caused by conditions inside the lumen, in the wall of the airway, as well as in the peribronchial region.

1. The lumen may be partially occluded by excessive secretions, for example by bronchial mucus (Tortora and Grabowski, 2000). This is a common cause of

chronic bronchitis. Partial obstruction can also occur acutely in pulmonary edema (fluid in the lungs) or after inhaling a foreign material.

2. Conditions in the airway wall include contraction of the bronchial smooth muscle (asthma), hypertrophy of the mucous glands (chronic bronchitis), and inflammation and edema of the wall (bronchitis and asthma).
3. Outside the airway, destruction of the lung parenchyma (alveolar tissue of the lung) can cause loss of radial traction and consequent narrowing, as in emphysema. A bronchus can compress locally by an enlarged lymph node or neoplasm, and peribronchial edema can also cause narrowing (West, 1998).

2.4.1.1 Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) is defined as a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema; the airflow obstruction is generally progressive, may be accompanied by airway hyperreactivity, and may be partially reversible (ATS, 1995). Diagnostic features are chronic productive cough, breathlessness on exertion, physiological evidence of airflow limitation, and poor reversibility (Woolcock, 1984; Snider, 1986). COPD affects about 30 million Americans and is the fourth leading cause of death following heart disease, cancer, and cerebrovascular disease (Tortora and Grabowski, 2000).

The most common cause of COPD is cigarette smoking or breathing secondhand smoke (Tortora and Grabowski, 2000). Cigarette smoke causes lesions that affect the small bronchi and bronchioles of the lungs. These lesions lead to inflammation and fibrosis that contributes to limitation in airflow. The predominant effect of smoking may be hypertrophy of mucous glands and hypersecretion, which can damage small airways,

destroy alveolar walls, and modify elastic recoil of the lungs (Fishman et al., 1998). Long-term studies of mortality clearly show a connection between cigarette smoking and death from bronchitis and emphysema (Taylor, 1978).

2.4.1.2 Emphysema

Emphysema is an abnormal permanent enlargement of the gas-exchanging units of the lungs in association with destruction of alveolar walls and without obvious fibrosis (ATS, 1962; Snider et al., 1985; ATS, 1995; Thurlbeck, 1995; Reid and Millard, 1964). The main physiological effect is a decrease in the elastic recoil of the lungs, which causes an outward displacement of the chest wall and flattening of the diaphragm, hyperinflation of the lungs, and increased resistance to airflow due to circumferential traction on the small airways by the over-distended lungs (Fishman et al., 1998). Hyperinflation places the respiratory muscles at a mechanical disadvantage, increasing the work and the oxygen cost of breathing (Fishman et al., 1998). Pulmonary function tests show a decrease in vital capacity (VC), forced expiratory volume in one second (FEV_1), maximal voluntary ventilation (MVV), and a reduced diffusing capacity in patients with emphysema (Frownfelter and Dean, 1996).

2.4.1.3 Chronic Bronchitis

Chronic bronchitis is a disease characterized by an excessive secretion of bronchial mucus, accompanied by a productive cough that is sufficient to cause an excessive rise of sputum (Tortora and Grabowski, 2000). These responses obstruct the airways and severely impair lung ventilation and gas exchange. Chronic bronchitis is not a single clinical entity, producing airflow limitation that leads to serious morbidity and mortality (Reid, 1960; Thurlbeck, 1995). Pulmonary function tests show a reduction in

VC, FEV_1, MVV, and diffusing capacity in patients with chronic bronchitis (Frownfelter and Dean, 1996).

2.4.1.4 Prognosis of Emphysema and Chronic Bronchitis

Chronic bronchitis and emphysema are associated with a progressive loss of lung function. Five years after being diagnosed with COPD, death rates are four to five times greater than normal values. Burrows and Earle (1969) reported an overall mortality of 47 percent at the end of five years. They believed that patients have an 80 percent survival rate with an FEV_1 greater than 1.2 L; 60 percent for those with an FEV_1 close to 1.0 L; and 40 percent with an FEV_1 less than 0.75 L. If the above rates are found in patients with complications of resting tachycardia, chronic hypercapnia and severally impaired diffusing capacity, the survival rates may be reduced by 25 percent (Frownfelter, 1987).

2.4.1.5 Treatment for Chronic Obstructive Pulmonary Disease

The focus in prevention of COPD has been on smoking cessation and the use of pharmacological agents, such bronchodilators and corticosteriods. Other treatments such as pulmonary rehabilitation have been used. Pulmonary rehabilitation may consist of patient education, nutritional assessments, exercise, breathing retraining, and psychological support (Ries, 1990; Celli et al., 1995). If pulmonary rehabilitation is unsuccessful, the patient will receive oxygen therapy (i.e., supplemental oxygen), and if previous rehabilitation attempts fail, then surgery such as a lung transplant is available.

2.4.2 Restrictive Diseases

During inspiration, the diaphragm descends, the chest wall moves upward and outward, and the lung expands as it fills with air. Therefore, an abnormality in any of these areas can produce a restrictive pattern. Restrictive diseases are those in which the

expansion of the lung is limited, either because of alterations in the lung parenchyma or because of disease of the pleura, the chest wall, or the neuromuscular apparatus (West, 1998). These diseases cause a reduction in vital capacity and resting lung volume, but airway resistance is not increased. The following section will expand on restrictive diseases.

2.4.2.1 Diseases of the Lung Parenchyma or Pleura

Diseases of the lung parenchyma such as interstitial fibrosis (thickened alveolar walls progressing to fibrosis) can decrease pulmonary compliance and elasticity, leaving the patient feeling fatigued, short of breath during exertion, and with a chronic unproductive cough (Frownfelter, 1987). Pleural abnormalities such as pleural effusion (movement of fluid into the pleural space) can prevent the lungs from expanding. Pleural effusion occurs in patients with congestive heart failure, pneumonia, etc. Symptoms may include chest pain, restriction of the chest wall, dyspnea, and fever (Frownfelter, 1987).

2.4.2.2 Diseases of the Chest Wall

The chest wall consists of the abdomen, the bony structure of the rib cage, the respiratory muscles, and the spine and its articulations. Diseases in any of the structures that make up the chest wall, by themselves or in combination with other diseases, interfere with ventilation and lead to respiratory failure (Fishman et al., 1998). A common disease of the chest wall that has a profound effect on pulmonary function is kyphoscoliosis, which produces the most restrictive pattern. Another disease that involves the bones of the rib or spinal column is ankylosing spondylitis and has much less of an impact on ventilatory capacity (Fishman et al., 1998).

2.4.2.2.1 Kyphoscoliosis

Kyphoscoliosis is characterized by a curvature of the spine laterally (scoliosis) and in the anteroposterior direction (kyphosis) (Frownfelter, 1987). These curvatures result in the vertebrae to rotate, forcing the ribs opposite of the curved side to bulge. The ribs of the concave side of the lateral curvature are sunken and depressed (Frownfelter, 1987) (Figure 2.3) Pulmonary function tests show a reduction in TLC and VC, with a relative preservation in RV. This decrease in TLC is a result of either a decrease in chest wall compliance or inspiratory muscle weakness.

2.4.2.2.2 Ankylosing Spondylitis

Ankylosing spondylitis is a rheumatic disease of unknown origin that attacks the axial skeleton. Chronic inflammation results in fibrosis and ossification of the ligamentous structure of the spine, sacroiliac joint, and rib cage. These inflammatory changes result in rigidity of the spine and loss of mobility of the rib cage due to bony ankylosis of the costovertebral and sternoclavicular joints (Fishman et al., 1998). As a result, the movement of the chest wall is decreased (West, 1998).

Clinically, ankylosing spondylitis is characterized by chronic low back pain with a decrease in spinal mobility and rib cage expansion. The rib cage moves less than 2.5 cm, at the level of the fourth intercostals space during a VC maneuver. Normal rib cage movement is greater than 2.5 cm at the level of the fourth intercostals space. Respiratory complaints such as dyspnea and chest wall pains are common and may be due to chest wall restriction. Some patients also experience mild exercise intolerance (Fishman et al., 1998).

2.4.2.2.3 Treatment of Kyphoscoliosis and Ankylosing Spondylitis

Medical therapies for adults with kyphoscoliosis and ankylosing spondylitis that are both preventative and supportive include immunization, adequate hydration, prompt treatment of respiratory infections, avoidance of sedatives, and carefully monitored supplemental oxygen. Since the degree of spinal deformity is associated with the development of respiratory failure in idiopathic kyphoscoliosis, surgical therapy has been used as an early intervention to stabilize the spine. Physical training should also be utilized to improve exercise tolerance.

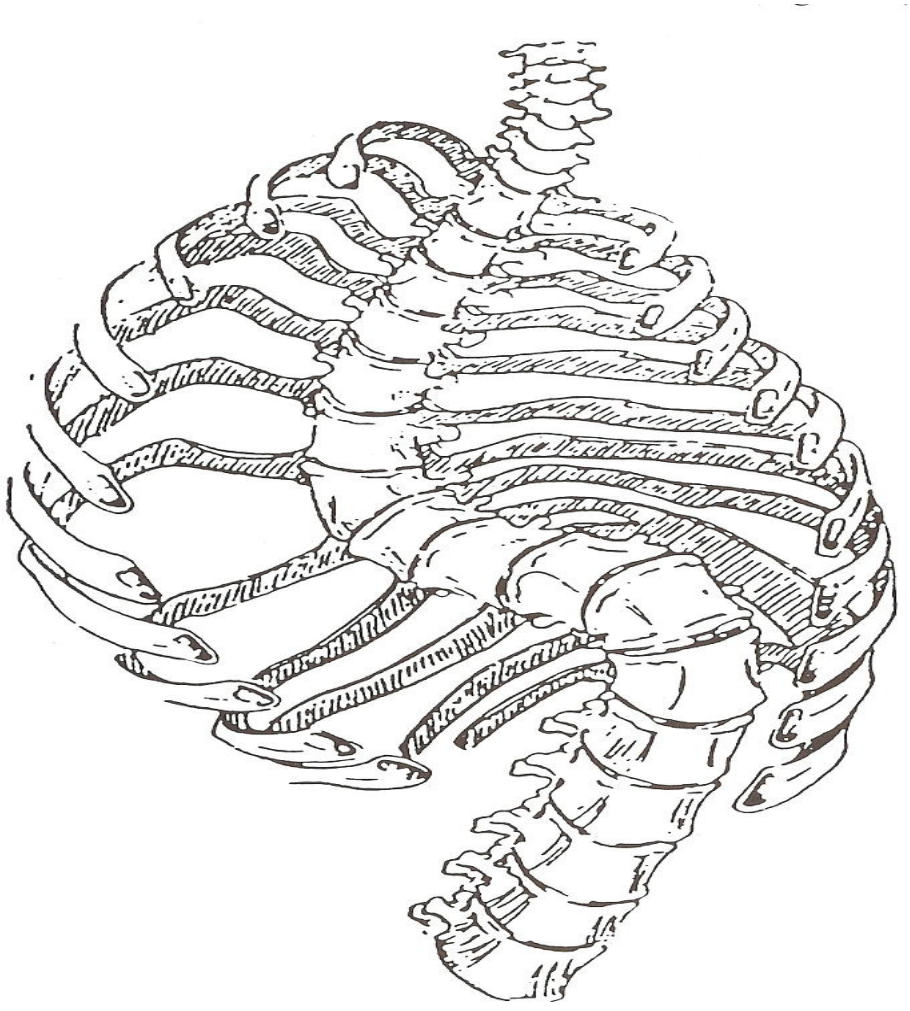

Figure 2.2. Schematic representation of the rotation of the spine and rib cage seen with scoliosis (Fishman et al., 1998).

2.4.2.3 Neuromuscular Disorders

Another classification of pulmonary diseases includes neuromuscular disorders (NMDs). NMDs are diseases that affect the respiratory muscles or the nerve supply to this area. The breathing pattern for patients with NMDs is often abnormal compared to healthy individuals. Patients with NMDs typically have respiratory muscle weakness, and resulting in a low tidal volume and a high respiratory rate (Baydur, 1991). The degree of ventilatory impairment among these disorders is dependent on the specific neuromuscular disease. Baydur (1991) showed that ventilatory output in NMD increased in spite of muscle weakness. However, rapid shallow breathing that is exhibited in these patients, is not due to abnormalities in gas exchange (Bautin et al., 2005) but more likely due to severe muscle weakness (Baydur, 1991) and/or disordered afferent and efferent output in motorneurons impaired by the neuromuscular disease (Begin et al., 1980).

Typical features seen in NMDs are a reduced FVC, decreased respiratory muscle strength, and, in some cases, malfunction of the neurons that control breathing. Patients with NMDs who develop respiratory muscle weakness may demonstrate fatigue, dyspnea, impaired control of secretions, recurrent lower respiratory tract infections, acute or chronic presentations or respiratory failure, and pulmonary hypertension. The pattern, prognosis, and degree of respiratory muscle weakness attributed to NMDs are varied. They depend on the level of neuromuscular system impairment, prognosis of underlying disorder, and whether therapy is available. Patients with Guillain-Barre syndrome tend to have less severe respiratory muscle weakness compared to patients with lower motoneuron lesions. Also, all respiratory muscles are not equally impaired; it depends on the course of the underlying NMD (Fishman et al., 1998).

2.4.2.3.1 Treatment of Neuromuscular Disorders

Principles for managing respiratory dysfunction in patients with neuromuscular disease include preventive therapies, such as respiratory muscle training, and stabilization of patients who develop acute or chronic presentations of respiratory failure. Inspiratory muscle aids that assist or support inspiratory effort are used along with expiratory muscle aids that assist in coughing.

2.5 Respiratory Muscle Fatigue

Studies of healthy individuals have indicated that the respiratory skeletal muscles, like muscles of the limbs, fatigue under conditions of intense activity, leading to respiratory failure (Bellemare and Grassino, 1982a; Bellemare and Grassino, 1982b; Cohen et al., 1982). Conditions that increase the level of inspiratory muscle activity or the duty cycle of breathing, or decrease the maximal pressure-generating capacity of the muscle, make fatigue more likely.

Respiratory muscle fatigue is defined as a loss in the capacity for developing force and/or velocity of a muscle resulting from muscle activity under load and is reversible by rest (NHLBI, 1990). Respiratory muscle weakness is impairment in the capacity of a fully rested muscle to generate force (NHLBI, 1990). Fatigue develops when the muscle is highly active and generating a substantial level of force. Muscle weakness is caused by muscle fiber atrophy, metabolic derangements that impair the ability of actomyosin cross-bridges to generate force, or chronic reductions in muscle precontraction length that impose a mechanical disadvantage (Fishman et al., 1998). Recovery from fatigue is generally observed over a short time (minutes to hours) versus recovery time for muscle weakness, which can take up to days if not weeks.

2.5.1 Classification of Respiratory Muscle Fatigue

Two classifications of respiratory muscle fatigue are central fatigue, failure of the central nervous system (CNS) to fully activate the respiratory muscles, and peripheral fatigue, where fatigue occurs within the muscle itself.

2.5.2 Measurements of the Diaphragm

Studies designed to examine the pathogenetic mechanisms that lead to respiratory muscle fatigue have focused mainly on the diaphragm. The diaphragm has been the primary focus of attention for many reasons. First, the diaphragm is the major respiratory muscle. Second, the anatomic considerations allow the mechanical output of the diaphragm (transdiaphragmatic pressure) and its electromyogram (EMG) to be assessed easily. Finally, the cervical nerves controlling the muscle can be electrically stimulated, allowing the mechanical output of the muscle to be assessed under normal conditions as well as during volitional contractions (Fishman et al., 1998).

A less invasive way of measuring transdiaphrgmatic pressure (Pdi) is by measuring maximal inspiratory mouth pressure (PI_{max}). Maximal inspiratory pressure reflects the combined strength of all the inspiratory muscles, and it is commonly measured as the maximal vacuum pressure that an individual can generate by inhaling against an occluded airway (Grassino and Clanton, 1991; Larson et al., 2002). With this maneuver, maximal force is difficult to assess because there are many inspiratory muscles activated, and the diaphragm can easily be uncoupled from the rib cage muscles during this maneuver and may be only partially active (De Troyer and Estenne, 1981; Laporta and Grassino, 1985).

Inspiratory muscle strength is determined by measuring the maximal inspiratory mouth pressure (PI_{max}) at RV and the maximal expiratory mouth pressure (PE_{max}) at TLC (Black and Hyatt, 1969). Inspiratory muscle endurance (peak pressure (P_m)) is the pressure achieved with the heaviest load until the subject is exhausted and can no longer inspire (Inbar et al., 2000).

2.5.3 Inspiratory Muscle Fatigue Among Populations with Pulmonary Diseases

Patients with moderate to very severe COPD see a reduction in functional strength of the inspiratory muscles (Sharp et al., 1968; Aldrich et al., 1982; Polkey et al., 1996). Inspiratory muscle strength usually is decreased to between 40 to 60 percent of predicted normal values, depending on the severity of the condition (Larson et al., 2002). Increasing airflow obstruction is accompanied by hyperinflation of the chest, placing the inspiratory muscles at a mechanical disadvantage (Decramer et al., 1994) and thus contributing to a decline in inspiratory muscle strength. The diaphragm becomes shorter and flattens, reducing its resting length and decreasing the maximal tension the diaphragm is able to generate (Kim et al., 1976; Ward et al., 1994). The reduction in force generated by the diaphragm produces a functional weakness of the inspiratory muscles, thus decreasing maximal inspiratory pressure (PI_{max}) (Larson et al., 2002).

Inspiratory muscle weakness can contribute to the restrictive process in patients with kyphoscoliosis (Smyth et al., 1984; Lisboa et al., 1985; Kearon et al., 1993) and ankylosing spondylitis (Vanderschueren et al, 1989). In young patients with mean scoliosis angles of about 50 degrees, PI_{max} and PE_{max} were decreased to 70 and 80 percent of control (Smyth et al., 1984; Kearon et al., 1993). In older adults with scoliosis, PI_{max} is about 50 percent of predicted (Lisboa et al., 1985). These reductions in maximal

inspiratory and expiratory strength may not be due to the intrinsic muscle disease but to the changes in the geometry of the chest wall affecting the mechanics of the inspiratory muscles (Lisboa et al., 1985).

Vanderschueren et al. (1989) reported a reduction in PI_{max} and PE_{max} to 76 ± 28 and 56 ± 17 percent of predicted values among patients with ankylosing spondylitis. They believed this was due to atrophy of the intercostal muscles resulting in diminished rib cage mobility and not diaphragm dysfunction.

Patients with NMDs, typically have a reduction in respiratory muscle strength compared to the normal population (Mulreany et al., 2003). Mulreany et al. (2003) showed that $PI_{max,}$ in patients with NMDs (43.0 ± 22.7 cm H_2O) was significantly lower than the healthy group (98.8 ± 20.7 cm H_2O). The main characteristic of chronic NMD is a decrease in VC. Reduction of VC is a consequence of respiratory muscle weakness. De Troyer (1980) found that reductions in VC were solely based on the reductions in inspiratory muscle strength, leading to a 40 percent decrease in lung compliance.

2.5.3 Inspiratory Muscle Fatigue Among Healthy Individuals

During high-intensity, exhaustive, constant-load cycling exercise above 85 percent VO_{2max} (Perret et al., 1999) or 80 percent of maximal work (W_{max}) (Bye et al., 1984), the diaphragms of healthy subjects can become fatigued. Perret et al. (1999) showed a significant decrease of 43 percent in respiratory muscle performance five minutes after cessation of exercise. After 0.5 to 2 minutes, Bye et al. (1984) showed a decrease in transdiaphragmatic pressure (Pdi) from 190 ± 26 to 167 ± 24 cm H_2O, and five minutes later Pdi was still at 169 ± 35 cm H_2O. These results correlate with the

findings of the diaphragm EMG during exercise, suggesting that diaphragmatic fatigue can occur in healthy individuals during exercise.

2.5.4 Inspiratory Muscle Fatigue Among Healthy Active Individuals

Inspiratory muscle fatigue has been demonstrated during high-intensity exercise in healthy active individuals (Perret et al., 2000) and triathletes (Boussana et al., 2001), and after completion of a marathon (Loke et al., 1982) or ultramarathon (Ker and Schultz, 1996). Perret et al. (2000) found breathing endurance was similarly reduced in constant-resistive breathing tests 10 minutes and 45 minutes after exhaustive cycling at either 65, 75, 85, or 95 percent VO_{2peak}. Boussana et al. (2001), reported a significant decrease in maximal inspiratory pressure (PI_{max}) (130 ± 3.8 to 126.7 ± 4.3 cm H_2O) among tri-athletes after 20 minutes of cycling followed by 20 minutes of running, and in a separate trial of 20 minutes of running followed by 20 minutes of cycling PI_{max} decreased from 129.6 ± 4.3 to 123.7 ± 4.9 cm H_2O. Loke et al. (1982), show decreases between pre- and post-race PI_{max} (from 165.8 ± 11.0 to 138.5 ± 7.6 cm H_2O), PE_{max} (from 240.0 ± 20.4 to 173.0 ± 22.6 cm H_2O), transdiaphragmatic pressure (from 78.8 ± 11.6 to 63.3 ± 7.0 cm H_2O) and MVV (from 178 ± 24.2 to 161.2 ± 23.2 L/min) following completion of a marathon race. These decreases in respiratory muscle strength and endurance suggest that the development of respiratory muscle fatigue after marathon running is possible. Ker and Shultz (1996) had their participants breathe to exhaustion against a constant resistive load before and after completion of an ultra-marathon. They demonstrated a reduction in endurance time of 26.5 percent, and this impairment was still evident three days after the race. Although the mechanisms for these changes are unclear,

it is likely that the development of respiratory muscle fatigue can be experienced with endurance exercise (Loke et al., 1982).

2.6 Respiratory Muscle Training

With a decrease in inspiratory muscle strength, inspiratory muscles must perform at a higher level during routine daily activities to maintain alveolar ventilation (Larson et al., 2002). The pressure developed with each breath is a greater percentage of the maximal pressure, thereby increasing vulnerability to fatigue (Larson et al., 2002). The negative relation between inspiratory muscle strength and fatigability of the respiratory muscles has been demonstrated in patients with pulmonary diseases and in healthy individuals after exercise training (Larson et al., 2002) (Respiratory Muscle Fatigue section 2.5). There is evidence that inspiratory muscle training (IMT) improves strength and endurance of the inspiratory muscles if the intensity of training adequate.

2.6.1 Respiratory Muscle Training Techniques

The potential role of respiratory muscle fatigue as a cause of respiratory failure and ventilatory limitation in patients with chronic pulmonary disease has stimulated attempts to train respiratory muscles (Smith et al., 1992). IMT is used to improve inspiratory muscle strength and endurance, with the ultimate goal of relieving symptoms of dyspnea and improving health-related quality of life in patients with pulmonary diseases. Three techniques have been used to train ventilatory muscles: (1) voluntary isocapnic hyperpnea (Boutellier and Piwko, 1992), (2) inspiratory resistive loading (Hanel and Secher, 1991; Gething et al., 2004), and (3) inspiratory threshold loading (Hanel and Secher, 1991; Suzuki et al., 1993; Inbar et al., 2000; Covey et al., 2001;

Voliantis et al., 2001; Williams et al., 2002; Larson et al., 2002). These training modes and influence upon respiratory muscle function are described below.

2.6.1.1 Voluntary Isocapnic Hyperpnea

The first technique to train ventilatory muscles is voluntary isocapnic hyperpnea (VIH). The patient is instructed to breathe at as high a level of minute ventilation as possible for up to 30 minutes (McConnell and Romer, 2004). This technique requires the patient to hyperventilate. Therefore, a rebreathing circuit that supplies supplemental O_2 must be used to maintain isocapnia (i.e., normal arterial carbon dioxide level) (Ehrman et al., 2003). Training sessions are typically conducted three to five times per week at approximately 60 to 90 percent of MVV. Using VIH, investigators have shown increases in the time to exhaustion during sustained isocapnic ventilation (Boutellier and Piwko, 1992; Boutellier et al., 1992), VC (Belman and Gaesser, 1988), maximal sustainable ventilatory capacity (Belman and Gaesser, 1988), and MVV (Leith and Bradley, 1976).

2.6.1.2 Inspiratory Resistive Loading

During inspiratory resistive loading, the patient breathes through inspiratory tubes of smaller and smaller diameter while attempting to maintain a normal breathing pattern (Ehrman et al., 2003). The smaller the diameter the greater the resistive load. Studies have reported increases in inspiratory muscle strength in the range of 18 to 54 percent (Leith and Bradley, 1976; Hanel and Secher, 1991). Leith and Bradley (1976) also observed small increases in TLC of about five percent.

2.6.1.3 Inspiratory Threshold Loading

With inspiratory threshold loading, the patient breathes through a device that only permits air to flow through it once a critical inspiratory pressure has been achieved

(Ehrman et al., 2003). Training with inspiratory threshold loading increases maximal velocity (Romer and McConnell, 2003), the maximal rate of shortening (Romer and McConnell, 2003), the maximal power output (Romer and McConnell, 2003), and endurance (Inbar et al., 2000) of the inspiratory muscles.

2.6.2 Inspiratory Muscle Training Among Diseased Populations

Patients with chronic COPD improve inspiratory muscle strength (Larson et al., 1999; Covey et al., 2001) and endurance (Larson et al., 1999) with the use of inspiratory muscle training by reducing the perceived effort of breathing and reducing the intensity of dyspnea associated with a given level of ventilation. These studies examined the effects of IMT on respiratory muscle performance in subjects with severe to very severe airflow obstruction (FEV_1 less than 65 percent of predicted and FEV_1/FVC less than 70 percent). Covey et al. (2001) demonstrated an increase in maximal inspiratory pressure (PI_{max}) (from 64 ± 15 cm H_2O to 75 ± 17 cm H_2O), peak pressure (P_m) (from 37 ± 12 cm H_2O to 53 ± 13 cm H_2O) and a reduction in dyspnea after four months of training. Larson et al. (1999) also reported an increase of 10 percent in inspiratory muscle strength, an increase of 28 percent in respiratory muscle endurance, and a decrease in dyspnea after training with cycle ergometry. However, improvements in exercise-related dyspnea did not indicate improvements in dyspnea during daily activities. To improve performance in a given activity one must train for that activity.

Lisboa et al. (1984) were the first to describe the technique of IMT with kyphoscoliotic patients. They found that PI_{max} was significantly lower in patients with kyphoscoliosis than in healthy subjects. With IMT, these patients showed significant increases in PI_{max} (from 42.3 ± 15.3 to 61.2 ± 16.5 cm H_2O). Hornstein et al. (1987)

published a case study describing a 26-year-old woman with idiopathic kyphoscoliosis and severe restrictive lung disease. The patient's main complaint was uncomfortable shortness of breath while climbing stairs and walking after a meal. After six months of IMT, PI_{max} and maximal expiratory pressure (PE_{max}) increased by 63 percent (from 38 to 62 mm Hg) and 70.9 percent (from 55 to 94 mm Hg) with an increased ability to walk and climb stairs with comfort. There were also increases in pulmonary functions.

Inspiratory and expiratory muscle training may improve respiratory muscle strength and endurance in patients with NMDs (DiMarco et al, 1985; McCool et al., 1986). Chest wall and pulmonary compliance are reduced in NMDs, increasing ventilatory workload in patients who already have a diminished ventilatory pump capacity. This imbalance between load and capacity of the respiratory muscles may lead to fatigue and respiratory failure (Rochester and Arora, 1983), ultimately causing death in some patients. Koessler et al. (2001) divided 27 subjects with NMDs into three groups based on VC values: group A (VC, 27 to 50 percent of normal predicted values), group B (VC, 51 to 70 percent of normal predicted values), and group C (VC, 71 to 96 percent of normal predicted values). After 24 months of resistive breathing, PI_{max} values significantly increased in group A from 51.45 ± 20.67 to 87.00 ± 12.73 cm H_2O, in group B from 59.38 ± 19.45 to 94.4 ± 29.94 cm H_2O, and in group C from 71.25 ± 22.87 to 99.00 ± 26.87 cm H_2O. It seems logical to use respiratory muscle training to improve inspiratory muscle function in order to delay the early onset of respiratory failure in patients with NMDs.

2.6.3 Inspiratory Muscle Training Among Healthy Individuals

Inspiratory muscle strength (Hanel and Secher, 1991; Suzuki et al., 1993) and endurance (Boutellier and Piwko, 1992) has been shown to increase among healthy individuals. Hanel and Secher (1990) showed a 32 percent increase in PI_{max} (from 93 mm Hg to 110 mm Hg), while Suzuki et al. (1993) showed a 34.9 percent increase in PI_{max} (from 98.3 ± 18.9 cm H_2O to 131.0 ± 21.2 cm H_2O) and a 15.6 percent increase in maximal expiratory pressure (PE_{max}) (from 127.3 ± 35.4 cm H_2O to 146.0 ± 37.2 cm H_2O) after four weeks of resistive and threshold breathing. Hanel and Secher (1990) also showed an eight percent increase in distance covered while running for 12 minutes on a track. Boutellier and Piwko (1992) demonstrated an increase in breathing endurance from 4.2 ± 1.9 to 15.3 ± 3.8 minutes after four weeks of voluntary isocapnic hyperpnea.

2.6.4 Inspiratory Muscle Training Among Active Individuals

Previous studies have demonstrated that respiratory muscles, like other skeletal muscles, respond to training by increasing strength (Inbar et al., 2000; Volianitis et al., 2001; Williams et al., 2002) and endurance (Inbar et al., 2000; Williams et al., 2002) in healthy active individuals. After 10 weeks, Inbar et al. (2000) reported a significant increase in PI_{max} from 142.2 ± 24.8 to 177.2 ± 32.9 cm H_2O and peak pressure (P_m) from 121.6 ± 13.7 to 154.4 ± 22.1 cm H_2O among well-trained endurance athletes. In fourteen female competitive rowers, Volianitis et al. (2001) demonstrated an increase in PI_{max} by 45.3 ± 29.7 percent (from 104 ± 8 to 148 ± 10 cm H_2O), with a reduction in respiratory muscle fatigue and an improvement in rowing performance after 11 weeks of IMT.

Williams et al. (2002) showed an increase in PI_{max} of 31 percent and an increase of 128 percent in breathing endurance time in seven college cross-country runners.

2.7 Chest Wall Restriction

Chest wall restriction reduces the ability of the chest wall to expand during inhalation and results in decreases in lung capacity (Caro et al., 1960; Stubbs and Hyatt, 1972; Bradley and Anthonisen, 1980; De Troyer, 1980; DiMarco et al. 1981; Klineberg et al., 1981; Scheidt et al., 1981; van Noord et al., 1986), resting pulmonary function (Cline et al., 1999; Gonzalez et al., 1999), and exercise performance (Vanmeenen et al., 1984; Hussain and Pardy, 1985; Hussain et al., 1985; O'Donnell et al., 2000; Coast and Cline, 2004). Chest wall restriction is observed in some forms of skeletal and pulmonary diseases (scoliosis) as well as occupational situations (Cline et al., 1999).

2.7.1 Pulmonary Diseases Restricting the Chest Wall

In diseases like ankylosing spondylitis and kyphoscoliosis, chest restriction is achieved through the deformities and inflammation of the spine, which decrease the expansion of the rib cage. Compared to other chest wall diseases, kyphoscoliosis produces the most severe restrictive pattern, producing a significant impact on pulmonary function. In nine patients with kyphoscoliosis, TLC and FVC are reduced to 43.6 ± 11.3 and 29.22 ± 7.53 percent, respectively, of predicted, with relatively no change in RV (94 ± 31.7 percent of predicted) due to inspiratory muscle weakness (Lisboa, et al., 1985). A decrease in TLC can also be due to a reduction in chest wall compliance. In adults, chest wall compliance may be reduced to only 20 to 30 percent of predicted (Fishman et al., 1998). Reductions in lung compliance are not as intense as reductions of chest wall compliance, but lung compliance also contributes to the restrictive process. Changes in

the lung compliance are a result of the static chest wall. Stiffening of the chest wall leads to a reduction in lung compliance by reducing FRC, resulting in a flatter portion of the volume pressure curve of the lung (Fishman et al., 1998).

Ankylosing spondylitis alters respiratory mechanics by severe restriction of rib cage mobility. The chest wall and total respiratory system compliance is reduced and chest wall resistance is increased. Although lung compliance decreases, TLC and VC are only mildly restricted (Feltelius et al., 1986; Vanderschueren et al., 1989). Feltelius et al. (1986) and Vanderschueren et al. (1989) demonstrated TLC at 92 and 85 percent and VC at 88 and 79 percent of normal values with reductions in pulmonary function. In patients with ankylosing spondylitis, on average TLC is reduced to 80 percent of predicted, depending on the severity of the spinal ankylosing. VC is also reduced to 70 percent of predicted, depending on the lack of rib cage expansion, disease activity and duration (Feltelius et al., 1986), and spinal mobility (Vanderschueren et al., 1989). Patients with ankylosing spondylitis tend to have lower pulmonary functions than normal subjects (Elliot et al., 1985).

2.7.1.1 Effects Kyphoscoliosis and Ankylosing Spondylitis has on Exercise

Exercise capacity in patients with mild to moderate kyphoscoliosis has been shown to be significantly less compared to that in normal individuals (Kearon et al., 1993). The lower the FVC, FEV_1 and MVV, the lower the VO_{2max} (Kesten et al., 1991). FVC and MVV have been shown to be 29.22 ± 7.53 and 37.5 ± 6.72 percent, respectively, compared to normal individuals (Lisboa et al., 1985). Keston et al. (1991) also showed that lung volumes in 15 adults with kyphoscoliosis were significantly lower than predicted values. VO_{2max} was significantly lower compared to predicted normal

values (31.60 ± 9.12 vs. 37.07 ± 4.91 $ml \cdot kg \cdot min^{-1}$). Depending on the severity of the scoliosis, VO_{2max} may be reduced to 60 to 80 percent of predicted. During exercise, patients with idiopathic thoracic scoliosis demonstrated an increased respiratory frequency (38 ± 8.6 bpm) compared to normal subjects (35 ± 8.8 bpm), with a decrease in tidal volume (Kearon et al., 1993).

Exercise capacity is mildly reduced in patients with ankylosing spondylitis and may in part be limited by inspiratory muscle performance. Elliot et al. (1985) suggests that reductions of VO_{2max} and work rate demonstrated by subjects with ankylosing spondylitis resulted from decreased quadriceps muscle strength rather than ventilatory limitation to exercise. Lack of evidence between the limitation of chest wall expansion and VO_{2max} or exercise tolerance suggests that exercise tolerance is not limited by the cardiac or respiratory system but by deconditioning (Keston et al., 1991).

2.7.2 Chest Restriction in Healthy Population

In healthy individuals, chest wall restriction can be achieved through emersion of water (Bradley and Anthonisen, 1980) or by wearing a non-elastic corset (Caro et al., 1960; Stubbs and Hyatt, 1972; Manco and Hyatt, 1975; Sybrecht et al., 1975; Klineberg et al., 1981; Scheidt et al., 1981; Vanmeenen et al., 1984; Hussain and Pardy, 1985; Hussain et al., 1985; van Noord et al., 1986), fiberglass shell with inflatable pads (Cline et al., 1999; Gonzalez et al., 1999), a belt (De Troyer, 1980), rubberized cloth bandages (DiMarco et al., 1981), wooden blocks (DiMarco et al., 1981), pneumatic cuff (Bradley and Anthonisen, 1980), or a weighted vest (Wang and Cerny, 2004).

In previous studies, chest restriction has shown to increase static elastic lung recoil or Pst (1) (Caro et al., 1960; Stubbs and Hyatt, 1972; Manco and Hyatt, 1975;

Sybrecht et al., 1975; De Troyer, 1980; Klineberg et al., 1981). Several mechanisms have been suggested as causes of this finding: (1) a reduction of the number of terminal lung units participating in ventilation (Caro et al., 1960), (2) a change in alveolar surface compliance (Stubbs and Hyatt, 1972; Sybrecht et al., 1975; De Troyer, 1980; Klineberg et al., 1981) (Stubbs 1972; Sybrecht 1975; De Troyer 1980; Klineberg 1981), and (3) a change in geometry of the thoracic cavity (Bradley and Anthonisen, 1978) with altered air space configuration.

2.7.2.1 Lung Volumes and Pulmonary Function

Chest restriction has been noted to decrease in VC by 38 percent, TLC 31.5 percent, FRC 31.5 percent and RV 13 percent (Caro et al., 1960; Stubbs and Hyatt, 1972; Manco and Hyatt, 1975; Sybrecht et al., 1975; De Troyer, 1980; Klineberg et al., 1981). Previous studies have shown decreases in pulmonary functions with chest restriction. Gonzalez et al. (1999) found decreased spirometric values resulting from chest wall restriction of 8, 11, and 10 percent in FVC, FEV_1, and MVV, respectively. Their results corroborate with the work by Cline et al. (1999), which reported decreases of 12 and 14 percent in FVC and FEV_1, respectively. Decreases in peak expiratory flow (PEF) (Stubbs and Hyatt, 1972; Klineberg et al., 1981) and an increase in maximal expiratory flow (MEFV) (Stubbs and Hyatt, 1972; Sybrecht et al., 1975; Klineberg et al, 1981) have been shown to be caused by chest wall restriction. Klineberg et al. (1981) displayed an increase in maximum expiratory flow volume (MEFV) from 3.22 ± 0.25 to 5.84 ± 0.69 L/s, while Stubbs and Hyatt (1972) showed increases from 3.2 to 6.0 L/s, and Sybrecht et al. (1975) from 3.6 to 4.8 L/s.

Chest wall restriction has been shown to increase respiratory frequency. Caro et al. (1960) and Stubbs and Hyatt (1972) reported an increase in respiratory frequency from 18 ± 1.8 to 23 ± 1.8 bpm and from 15 to 19 bpm, respectively, and a decrease in tidal volume, which has been seen in idiopathic thoracic scoliosis patients (Kearon et al., 1993), while MVV was essentially unchanged.

2.7.2.2 Elastic Lung Recoil

With chest wall restriction, the static pressure volume (PV) curves of the lung were less steep and shifted to the right (Caro et al., 1960; Stubbs and Hyatt, 1972; Sybrecht et al., 1975; De Troyer, 1980; Klineberg et al., 1981) thus reflecting an increase in elastic recoil pressure and a reduced pulmonary compliance. In previous studies, elastic lung recoil pressure increased from 4.99 to 7.78 cm H_2O, on average (Caro et al., 1960; Stubbs and Hyatt, 1972; Manco and Hyatt, 1975; Sybrecht et al., 1975; De Troyer, 1980; Klineberg et al., 1981). After release of the chest strap, the pressure-volume curve did not return to normal until the first deep breath to control TLC (Caro et al., 1960; Sybrecht et al., 1975; Bradley and Anthonisen, 1980).

Chest restriction has been shown to decrease lung and chest wall compliance. De Troyer (1980) showed a decrease in lung and chest wall compliance from 0.0463 to 0.373 cm H_2O and from 0.229 to 0.106 cm H_2O. At 50 percent TLC, Klineberg et al. (1981), Stubbs and Hyatt (1972), and Sybrecht et al. (1975), showed a decrease in lung compliance from 0.38 ± 0.04 to 0.23 ± 0.04 cm H_2O, from 0.426 to 0.287 cm H_2O, from 0.372 to 0.211 L/cm H_2O, respectively. De Troyer (1980) explains that despite reductions in compliance of both lung and chest wall, the total work required to overcome the elastic

forces during inspiration is increased by only 10 percent during strapping compared to the control state.

2.7.2.3 Changes in Alveolar Surface Compliance

Caro et al. (1960) first suggested that terminal lung units might close during chest restriction by demonstrating the release of small amounts of trapped gas after removing the chest restrictor. Manco and Hyatt (1975) tried replicating this and were unable to demonstrate gas trapping as a result of chest strapping. Caro et al. (1960) demonstrated a decreased in arterial oxygen tension and could not determine if this was due to an increase in intrapulmonary shunting (atelectasis) or to a reduction of mixed venous oxygen tension or to both. Klineberg et al. (1981) showed no significant correlation between the increase in lung recoil pressure and the increase in right-to-left intrapulmonary shunt.

2.7.2.4 Muscle Activity

Ventilatory loads applied at the mouth interfere with the movement of all the respiratory muscles and elicit increases in respiratory muscle activity (Altose et al., 1979). An application of a chest wall restriction device, which decreases its expansion, elicits increases in inspiratory muscle force (DiMarco et al., 1981). DiMarco et al. (1981) used occlusion pressure to determine muscle responses during unhindered or restricted breathing. They reported that the chest restriction group had an occlusion pressure of 251.9 ± 20.8 percent of the control group. The response to rib cage restriction is graded so that the greater the restriction the greater the increases in muscle activity (DiMarco et al., 1981). This was based on the observation that, in seated human subjects, rib cage tidal volume generally exceeds diaphragm tidal volume (Sharp et al., 1975), and increases in

ventilation are generally associated with increases in rib cage movements due to the progressive recruitment of the inspiratory intercostals (scalenes and sterncleidomastoids) (Macklem et al., 1977).

2.7.2.5 Effects of Chest Wall Restriction on Exercise

Previous studies have demonstrated that chest restriction decreases lung capacity and pulmonary function during exercise. These reductions led to decreases in respiratory muscle function (Hussain and Pardy, 1985; Hussain et al., 1985; Harty et al., 1999; O'Connor et al., 2000; O'Donnell et al., 2000; Coast and Cline, 2004; Wang and Cerny, 2004) and exercise performance (Vanmeene et al., 1984; Hussain and Pardy, 1985; Hussain et al., 1985; O'Donnell et al., 2000; Coast and Cline, 2004).

As mentioned earlier, application of chest wall restriction results in significant changes in resting lung volumes. Decreases in resting lung volumes result in rapid shallow breathing during exercise (Vanmeenen et al., 1984; Hussain and Pardy, 1985; Hussain et al., 1985; Harty et al., 1999; O'Connor et al., 2000; O'Donnell et al., 2000; Coast and Cline, 2004; Wang and Cerny, 2004). Coast and Cline (2004) showed a decrease in VO_{2max}, time to maximum exercise, maximum minute ventilation, and maximum tidal volume. Harty et al. (1999) reported that the experimental group had a significantly greater minute ventilation (61 vs. 49 L/min) and respiratory frequency (43 vs. 23 bpm) during exercise, compared to the control group. Hussain et al. (1985) illustrated that chest restriction reduced exercise time (T_{lim}) from 6.8 ± 0.7 to 4.3 ± 0.4 minutes, decreased tidal volume, increased breathing frequency by reducing inspiratory and expiratory time, and decreased diaphragmatic contraction, thus concluding that chest restriction alters the pattern of breathing in normal subjects during

high-intensity exercise. Hussain and Pardy (1985) reported significant reductions in diaphragmatic fatigue, resulting in an increase in exercise time from about seven minutes to four minutes at a constant workload of 80 percent of maximum power output. O'Connor et al. (2000) showed decreases in ventilation (from 103.7 ± 7.8 to 81.9 ± 4.3 L/min), tidal volume (from 2.6 ± 0.1 to 1.9 ± 0.2 Liters), VO_{2max} (from 43.8 ± 2.1 to 35.6 ± 2.4 $ml \cdot min^{-1} \cdot kg^{-1}$), and an increase in breathing frequency (from 38.6 ± 2.6 to 47.7 ± 3.7 bpm) during maximal exercise. O'Donnell et al. (2000) reported that chest restriction significantly reduced the tidal volume response to exercise, and increased dyspnea at any given work rate or ventilation, thus limiting exercise performance by 28 ± 3 percent. Vanmeenen et al. (1984) showed a reduction in maximal work capacity to 89 percent, in maximal minute ventilation by 14 percent, and tidal volume by 27 percent, while breathing frequency was increased by 14 percent. Wang and Cerny (2004) used chest restriction to simulate the effect of moderate obesity during exercise. The peak work rate achieved during exercise was lower with chest restriction compared to the control (121.88 ± 8.84 vs. 146.88 ± 8.84 W).

2.8 Effects of Aerobic Training on Aerobic Capacity

Aerobic exercise is the ability of the heart, lungs, and blood vessels to deliver an adequate supply of oxygen while exercising large muscle mass, performing rhythmic activity, walking, jogging, biking, swimming, etc. (Cotton, 1996). With aerobic training, the body adapts through alterations of physiological processes or systems, such as an increase in VO_{2max}, a decrease in resting heart rate (HR), a decrease in exercise HR at a given workload, no change or slight decrease in maximum heart rate, an increase in arterial-venous oxygen difference (A-VO_2), an increase in cardiac output, no change or

slight increase in systolic blood pressure, and an increase in oxidative capacity of muscle. The overall adaptation to aerobic exercise results in less effort by all organs at every possible level of exercise. The acute and long-term effects of aerobic exercise on the respiratory system have been studied extensively over the years.

2.8.1 Respiratory Changes to Aerobic Training

2.8.1.1 Acute Responses During Aerobic Training

During exercise, the respiratory system makes adjustments to both intensity and duration of the exercise. The ventilatory response to constant-load exercise is characterized by three distinct phases (Cunningham, 1974). The first phase, at the start of exercise, usually occurs within the first breath (Jensen et al., 1971). The second phase occurs after approximately 15 seconds with a time constant of 60 to 70 seconds (Broman and Wigertz, 1971). At phase three, ventilation reaches a constant level within approximately 4 minutes, if exercise is performed below the individual's anaerobic threshold (Wasserman et al., 1986). Above anaerobic threshold, ventilation will continue to slowly increase during phase three (Linnarsson, 1974) because of the disproportionate rise in CO_2. The abrupt increase in ventilation at the beginning of exercise is due to neural changes that send excitatory impulses to the inspiratory area of the medulla oblongata in the brain. These changes include the following (1) psychic stimuli (anticipation of the activity), (2) simultaneous cortical motor activation of the skeletal muscles and respiratory centers, and (3) excitatory impulses reaching respiratory centers from propriocepters in moving muscles, tendons, and joints (Marieb, 2004).

When exercise stops, an initial small but abrupt decline in ventilation rate occurs, followed by a gradual decrease to the pre-exercise value. This reflects the shutting off of

the neural control mechanisms. The gradual decline to baseline ventilation reflects a decline in CO_2 flow that occurs as the oxygen debt is being repaid (Marieb, 2004).

2.8.1.2 Chronic Adaptations to Aerobic Training

Changes in the respiratory system include increases in VO_{2max} (Daniels et al., 1978; Melanson et al., 1996; Orngreen et al., 2005) and lung volumes (Kaufmann and Swenson, 1981; Robinson and Kjeldgaard, 1982). Common symptoms of myotonic dystrophy are respiratory muscle weakness and atrophy that can become severe. Patients often report intolerance to exercise, which often leads to a sedentary lifestyle. Orngreen et al. (2005) studied 12 patients with myotonic dystrophy with complaints of physical weakness and fatigue. The study consisted of 12 weeks of aerobic training on a cycle ergometer at 65 percent of VO_{2max}. Training improved VO_{2max} by 14 percent (from 32.5 ± 3.8 to 37.3 ± 4.6 $ml \cdot min^{-1} \cdot kg^{-1}$) and maximal workload by 11 percent (from 154 ± 19 to 171 ± 22 W). Physical weakness and fatigue during activities of daily living improved with training. Aerobic training safely improves cardiovascular fitness, endurance, and self-assessed activities of daily living.

Continuous aerobic exercise can improve VO_{2max} (Daniels et al., 1978; Melanson et al., 1996) with improvements in pulmonary function (Kaufmann and Swenson, 1981; Robinson and Kjeldgaard, 1982) in healthy individuals. Daniels et al. (1978) demonstrated a significant increase in VO_{2max} after eight weeks of aerobic training. Within the first four weeks of training, VO_{2max} increased significantly but failed to increase further, even with an increase in training load. Melanson et al. (1996) reported that after nine weeks of aerobic training, either by running or in-line skating, significant

increases in VO_{2max} were observed by runners (46.3 ± 1.8 to 50.8 ± 1.9 $ml \cdot min^{-1} \cdot kg^{-1}$) and in-line skaters (46.1 ± 1.7 to 48.9 ± 1.7 $ml \cdot min^{-1} \cdot kg^{-1}$).

Kaufmann and Swenson (1981) studied two marathon runners who averaged 45 to 70 miles per week over three years. Marathon runner A demonstrated increases of TLC (from 8.42 to 8.60 L), FRC (from 3.68 to 5.17 L), FRC/TLC ratio (from 44 to 60 percent), and alveolar-capillary permeability (from 3.96 to 5.07 min^{-1}). Marathon runner B had similar increases in FRC (from 3.00 to 3.21 L), FRC/TLC ratio (from 46 to 54 percent), and alveolar-capillary permeability (from 4.75 to 5.17 min^{-1}). Robinson and Kjeldgaard (1982) reported significant increases in MVV (13.6 percent), maximum sustainable ventilatory capacity (15.8 percent), peak inspiratory flow (PIF), and PE_{max} after 20 weeks of aerobic training.

Any skeletal muscle in the body can be made to fatigue. This includes muscle that aid in breathing, such as the diaphragm and other accessory ventilatory muscles. Respiratory muscle fatigue can be delayed by improving respiratory muscle strength and endurance with IMT and exercise. By adding chest wall resistance using an externally applied thoracic constriction band around the chest during aerobic exercise, may result in improvements in aerobic capacity, which is discussed in the next sections.

CHAPTER 3

METHODS

3.1 Participants

Twenty-two healthy, non-active adults (26.95 ± 4.8 yrs (mean ± SD)) volunteered for the study. Participants read and signed an informed consent form prior to the commencement of the study (Appendix A). Participants completed a Physical Activity Readiness Questionnaire (Par-Q) (Appendix B) (CSEP, 2002). Pre-, mid-, and post-measurements were conducted as part of the study. Pre- and post-testing took place the week prior to and the week following the eight-week study period. Mid-testing was at week four. All stages of testing included a pulmonary lung function test and a VO_{2max} treadmill test.

3.1.1 Training Category

Following pre-testing, healthy non-active participants were randomly placed into one of two groups: (1) Chest Wall Restriction Group (CWR) (n=11), or (2) control group (Non Chest Wall Restriction Group (NCWR)) (n=11). Subjects in the CWR had their chest wall restricted by an elastic strap.

3.2 Procedures

3.2.1 Aerobic Training Program

All groups performed aerobic exercise three days a week for approximately 30 minutes at a moderate intensity estimated at 65 to 80 percent of their maximal exertion, with at least one day of rest between exercise sessions. Participants entered the study from sedentary life styles, thus exercise intensity was generally described as being winded but still able to hold a conversation. During the first two weeks of the training

program, sessions may have been as short as 15 minutes depending on individuals' aerobic capacity and ability to adapt to physical activity. The CWR Group performed the exercise sessions while wearing an elastic strap. Participants were encouraged to use a treadmill, elliptical machine, and/or stationary cycle randomly and in any order to complete the exercise session. Participants were also encouraged to complete an exercise journal, recording the duration and mode of aerobic exercise during each session (Appendix C).

3.2.2 VO_{2max} Testing

Participants' VO_{2max} levels were assessed using the Thoden Treadmill Protocol. Participants had a 3 to 5 minute warm-up at 3.0 mph and 0 percent grade prior to the start of the test. The Thoden Treadmill Protocol consisted of stages lasting two minutes each. The speed throughout the test was a static 6.0 mph for females and 7.0 mph for males, while the grade started at zero percent and gradually increased by two percent every two minutes (Thoden, 1991). The test continued until the participant reached complete physical failure or stopped at volitional request. The Quinton Q Treadmill, Seattle, WA (#1860), the PhysioDyne Instrument metabolic cart with a Max II oxygen analyzer (#Pm1111E), and a carbon dioxide analyzer (#1r1507) were used. Participants wore a Polar HR A1 heart rate monitor, Lake Success, NY (#190269), head support gear (#2726), and a Hans-Randolph two-way mouth piece (#2700, #7900, #2600, #1410) throughout the test.

3.2.3 Pulmonary Lung Function

Pulmonary lung function tests were performed with the subject standing. Participants breathed through a disposable pneumotach mouthpiece attached to the handle

of an IQmark Digital Spirometer, Torrance, CA (#521999). While breathing through the mouthpiece with a nose clip in place, the subjects performed forced vital capacity (FVC) loop (flow volume loop) maneuvers (after full inspiration, participants expired forcefully for six seconds, followed by a deep inspiration) until three reproducible measurements were recorded and stored on a computer for later analysis. Chest measurements at peak inspiration and expiration were also recorded.

3.2.4 Chest Constriction

Each subject in the CWR group had their chest wall restricted with the use of an elastic strap made by Mueller, Prairie du sac, WI (widths ranging from 10 to 15 cm) adjusted to fit just beneath the axillae and around the chest to envelop the rib cage. The desirable degree of lung restriction was achieved by manually tightening the straps. A 10 percent reduction in FVC from baseline was considered the target restriction. It is worth noting that this apparatus does not prevent the chest wall from expanding like earlier studies, but offers elastic concentric resistance for the inspiratory muscles to work against.

3.2.5 Statistical Analyses

Data analyses were conducted for each variable. MANOVAs with repeated measures (baseline, midpoint, endpoint) were applied for between-group comparisons where there was more than one related measure. ANOVAs with repeated measures were applied for individual groups where the outcome measures were single parameters. Where significant interactions were found for any of the measures, post-hoc analyses were conducted using the L-S-D and Tukey method to locate the means that were significantly different. This included within-subject comparisons of baseline to midpoint

(effects of first four weeks of exercise) and within-subject comparisons of midpoint to endpoint (effects of exercise from weeks four to eight). The repeated measures MANOVAs and ANOVAs were performed using SPSS (Version 12, SPSS Inc. Headquarters, Chicago, Illinois, USA). Data are expressed as the mean ± SD, and the level of significance was set at $P \leq 0.05$ for all variables.

CHAPTER 4

RESULTS

4.1 Subjects

Twenty-two, healthy non-active 18 to 35 year-old participants participated in the eight-week study. Participants were then randomly assigned to one of two groups: Chest Wall Restriction Group (CWR; n=11; male=6, female=5) or Control the Non-Chest Wall Restriction Group (NCWR; n=11; male=4, female=7) or control group. All participants in the CWR completed the study. One participant in the NCWR Group did not complete the study; this individual withdrew at week four due to pregnancy and her data were not used in the results. Another, subject did not participate in the spirometry testing at week four and week eight due to fear of inducing an asthma attack. Baseline and eight-week anthropometric data for the subjects in both groups are shown in Table 4.1 and 4.2.

TABLE 4.1

BASELINE ANTHROPOMETRIC DATA FOR CWR AND NCWR

Group	Age (yr)	Height (cm)	Weight (kg)
CWR (n=11)	27.00 ± 2.97	174.36 ± 10.34	79.55 ± 11.40
NCWR (n=10)	26.90 ± 6.49	172.20 ± 6.97	85.50 ± 17.55

TABLE 4.2

8-WEEK ANTHROPOMETRIC DATA FOR CWR AND NCWR

Group	Age (yr)	Height (cm)	Weight (kg)
CWR (n=11)	27.00 ± 2.97	174.36 ± 10.34	78.79 ± 11.51
NCWR (n=10)	26.90 ± 6.49	172.20 ± 6.97	85.41 ± 17.61

4.2 VO_{2max}

A significant interaction was observed between groups, representing a difference in VO_{2max} scores as participants were tested from baseline to midpoint to endpoint ($P < 0.05$). Follow-up post-hoc investigation indicated that the experimental group had a significant improvement in VO_{2max} after four weeks of exercise (from 33.55 ± 6.48 to 37.39 ± 6.68 $ml.min^{-1}.kg^{-1}$ ± SD) compared to the control group (from 33.30 ± 10.39 to 34.07 ± 10.14 $ml.min^{-1}.kg^{-1}$ ± SD) (Figure 4.1). At eight weeks experimental and control groups had significant increases compared to baseline in VO_{2max} from 33.55 ± 6.48 to 37.78 ± 7.11 and 33.30 ± 10.39 to 35.99 ± 9.09 $ml.min^{-1}.kg^{-1}$ ± SD, respectively (Table 4.3), with no differences between groups. However, a significant improvement (11.0 ± 4.0 percent) in aerobic capacity was observed in the experimental group at just four weeks, compared to the control group (3.0 ± 6.0 percent). There was no difference from week four to week eight. VO_{2max} values for each group can be seen in Tables 4.4 and 4.5.

TABLE 4.3

VO_{2max} VALUES ($ml \cdot min^{-1} \cdot kg^{-1}$) (mean ± SD) FOR CWR AND NCWR GROUPS AT BASELINE, 4, AND 8 WEEKS

Group	VO_{2max} Baseline	VO_{2max} 4 Weeks	VO_{2max} 8 Weeks
CWR (n=11)	33.55 ± 6.48	37.39 ± 6.68*	37.78 ± 7.11*
NCWR (n=10)	33.30 ± 10.39	34.07 ± 10.14	35.99 ± 9.09*

* $P<0.05$ from baseline to week 4 and week 8

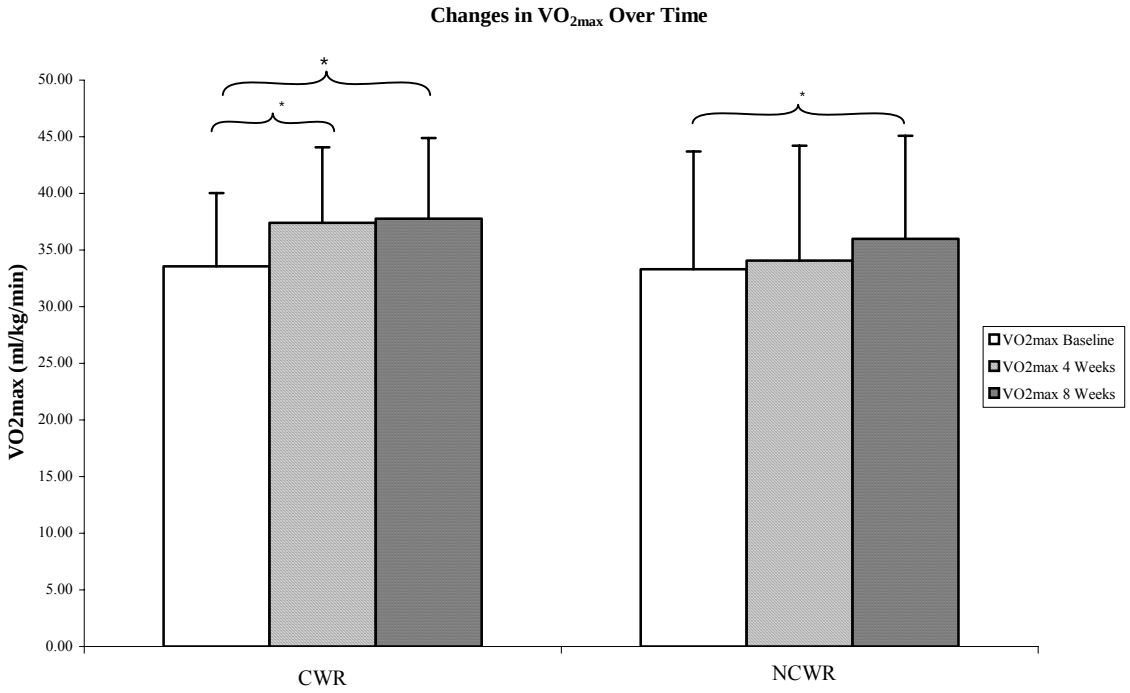

Figure 4.1. Change in aerobic power (VO_{2max}) (baseline, midpoint and endpoint). Data are presented as mean ± SD.
* $P < 0.05$ for within subject comparison.

TABLE 4.4

VO_{2max} VALUES ($ml \cdot min^{-1} \cdot kg^{-1}$) FOR CWR GROUP AT BASELINE, 4, AND 8 WEEKS

CWR Group	VO_{2max} Baseline	VO_{2max} 4 Weeks	VO_{2max} 8 Weeks
01	37.5	38.6	39.0
02	34.2	36.9	35.5
03	30.6	35.2	35.0
04	30.1	34.0	33.1
05	41.0	46.3	46.3
06	43.4	47.8	51.7
07	33.6	35.5	36.1
08	38.0	44.3	45.3
09	25.9	30.9	30.6
10	33.5	36.5	34.0
11	21.2	25.3	29.0

TABLE 4.5

VO_{2max} VALUES ($ml \cdot min^{-1} \cdot kg^{-1}$) FOR NCWR GROUP AT BASELINE, 4, AND 8 WEEKS

NCWR Group	VO_{2max} Baseline	VO_{2max} 4 Weeks	VO_{2max} 8 Weeks
01	28.0	28.0	30.3
02	48.7	49.1	49.6
03	27.7	29.1	30.3
04	45.8	47.5	50.0
05	43.5	39.7	36.7
06	21.1	24.5	29.9
07	23.3	23.1	25.7
08	26.7	27.2	30.1
09	41.9	45.1	45.9
10	26.3	27.4	31.4

4.3 Pulmonary Functions

No differences were found in FVC, FEV_1, $FEF_{25-75\%}$, and PEF ($P > 0.05$).

4.3.1 Forced Vital Capacity

The effects of aerobic training on FVC for both groups are listed in Table 4.6. No change in FVC in either group during the eight weeks of training in was seen (Figure 4.2). Individual FVC values for each group are listed in Tables 4.7 and 4.8.

TABLE 4.6

FORCED VITAL CAPACITY VALUES FOR CWR AND NCWR GROUPS AT BASELINE, 4, AND 8 WEEKS

Group	FVC (L) Baseline	FVC (L) 4 Weeks	FVC (L) 8 Weeks
CWR (n=11)	4.86 ± 1.21	4.77 ± 1.06	4.80 ± 1.10
NCWR (n=10)	5.15 ± 1.03	4.81 ± 1.30	4.92 ± 0.88

Values are shown as mean ± SD

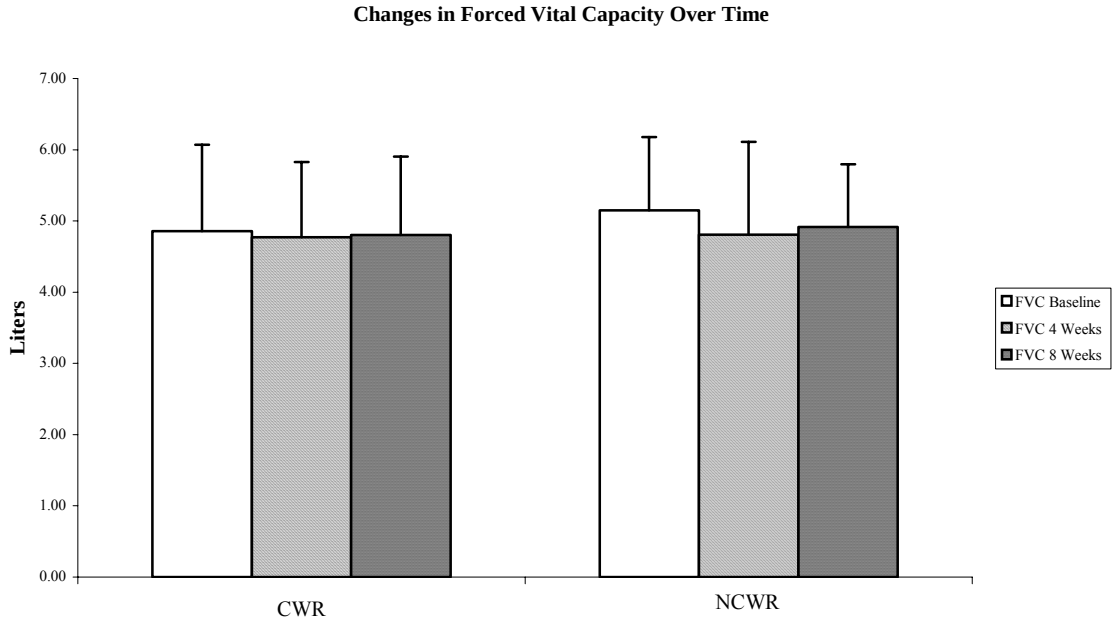

Figure 4.2. Forced vital capacity determined during spirometry test over eight-week period.
Data are presented as mean ± SD.

TABLE 4.7

FORCED VITAL CAPACITY VALUES FOR CWR GROUP AT BASELINE, 4, AND 8 WEEKS

Subjects	FVC (L) Baseline	FVC (L) 4 Weeks	FVC (L) 8 Weeks
01	7.175	5.867	5.679
02	6.439	5.934	6.499
03	4.252	3.852	3.786
04	4.277	4.126	3.773
05	4.415	4.145	4.382
06	4.554	5.799	5.759
07	3.942	4.076	4.311
08	5.842	6.140	6.090
09	3.721	3.502	3.626
10	5.483	5.474	5.417
11	3.314	3.579	3.522

TABLE 4.8

FORCED VITAL CAPACITY VALUES FOR NCWR GROUP AT BASELINE, 4, AND 8 WEEKS

Subjects	FVC (L) Baseline	FVC (L) 4 Weeks	FVC (L) 8 Weeks
01	3.692	4.056	3.797
02	5.909	3.643	4.881
03	4.033	4.020	4.054
04	5.942	5.164	5.419
05	6.541	6.861	5.834
06	4.523	N/A	N/A
07	4.087	2.885	3.932
08	5.071	4.939	4.807
09	6.340	6.455	6.365
10	5.347	5.257	5.148

4.3.2 Forced Expiratory Volume in One Second

The effects of aerobic training on FEV_1 for both groups are listed in Table 4.9. No significant change in FEV_1 in either group over the eight weeks of training was seen (Figure 4.3). Individual FEV_1 values for each group are listed in Tables 4.10 and 4.11.

TABLE 4.9

FORCED EXPIRATORY VOLUME IN ONE SECOND VALUES FOR CWR AND NCWR GROUPS AT BASELINE, 4, AND 8 WEEKS

Group	FEV_1 (L) Baseline	FEV_1 (L) 4 Weeks	FEV_1 (L) 8 Weeks
CWR (n=11)	3.78 ± 0.89	3.77 ± 0.63	3.76 ± 0.72
NCWR (n=10)	3.87 ± 0.75	3.86 ± 1.04	3.92 ± 0.62

Values are shown as mean ± SD

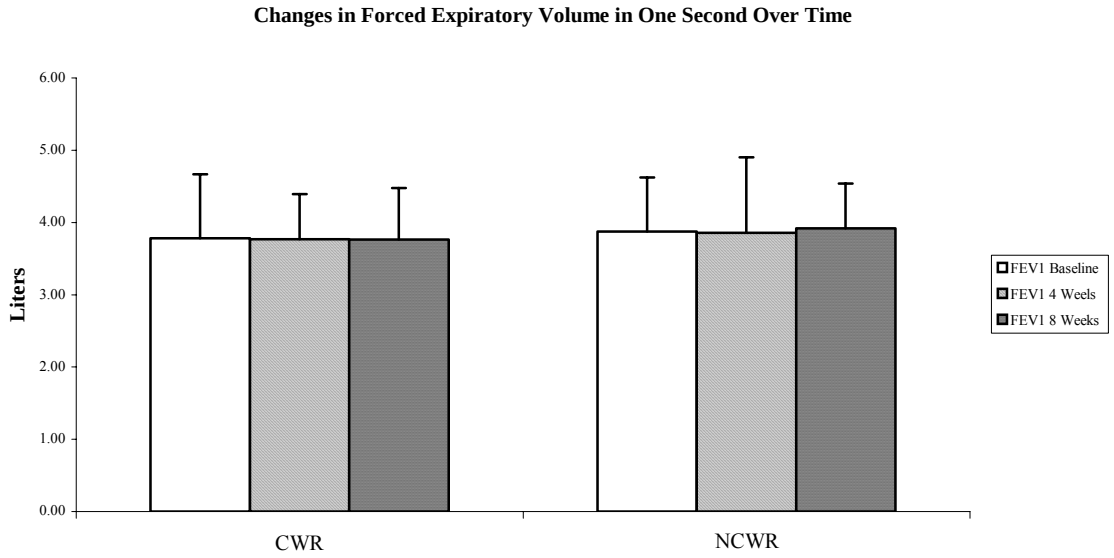

Figure 4.3. Forced expiratory volume in one second determined during spirometry test over eight-week period.
Data are presented as mean ± SD.

TABLE 4.10

FORCED EXPIRATORY VOLUME IN ONE SECOND VALUES FOR CWR GROUP AT BASELINE, 4, AND 8 WEEKS

Subjects	FEV_1 (L) Baseline	FEV_1 (L) 4 Weeks	FEV_1 (L) 8 Weeks
01	5.855	4.489	4.606
02	4.872	4.578	4.938
03	3.398	3.395	3.33
04	3.106	3.333	3.172
05	3.601	3.473	3.411
06	3.413	4.426	4.460
07	3.519	3.694	3.541
08	4.082	4.361	4.296
09	2.926	2.707	2.758
10	3.861	3.859	3.839
11	2.948	3.113	3.034

TABLE 4.11

FORCED EXPIRATORY VOLUME IN ONE SECOND VALUES FOR NCWR GROUP AT BASELINE, 4, AND 8 WEEKS

Subjects	FEV_1 (L) Baseline	FEV_1 (L) 4 Weeks	FEV_1 (L) 8 Weeks
01	3.096	3.615	3.347
02	3.441	2.918	4.023
03	3.019	3.096	3.147
04	4.718	4.061	4.181
05	5.373	5.965	4.818
06	3.447	N/A	N/A
07	3.384	2.412	3.262
08	4.019	3.811	3.603
09	4.161	4.399	4.772
10	4.077	4.433	4.107

4.3.3 Forced Expiratory Flow From 25 to 75 Percent

The effects of aerobic training on $FEF_{25-75\%}$ for both groups are listed in Table 4.12. No significant change in $FEF_{25-75\%}$ in either group over the eight weeks of training in was seen (Figure 4.4). Individual $FEF_{25-75\%}$ values for each group are listed in Tables 4.13 and 4.14.

TABLE 4.12

FORCED EXPIRATORY FLOW FROM 25 TO 75 PERCENT VALUES FOR CWR AND NCWR GROUPS AT BASELINE, 4, AND 8 WEEKS

Group	$FEF_{25-75\%}$ (L/s) Baseline	$FEF_{25-75\%}$ (L/s) 4 Weeks	$FEF_{25-75\%}$ (L/s) 8 Weeks
CWR (n=11)	3.67 ± 1.24	3.67 ± 0.77	3.54 ± 0.66
NCWR (n=10)	3.50 ± 0.82	3.83 ± 1.32	3.86 ± 0.72

Values are shown as mean ± SD

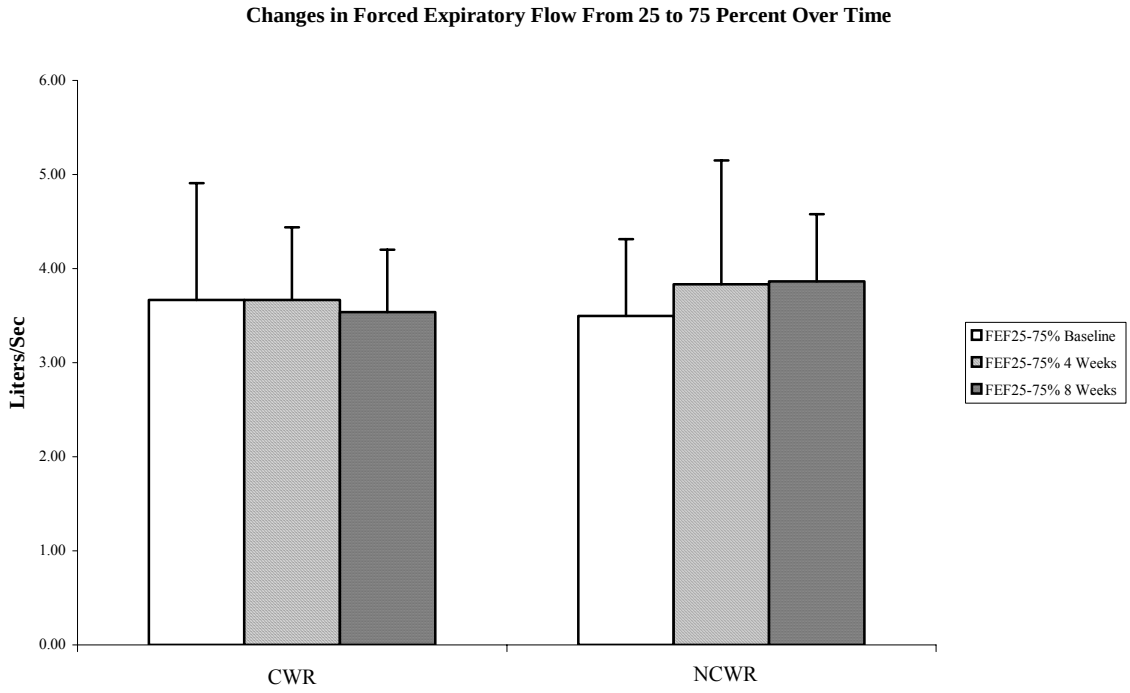

Figure 4.4. Forced expiratory flow from 25 to 75 percent determined during spirometry test over eight-week period.
Data are presented as mean ± SD.

TABLE 4.13

FORCED EXPIRATORY FLOW FROM 25 TO 75 PERCENT VALUES FOR CWR GROUP AT BASELINE, 4, AND 8 WEEKS

Subjects	$FEF_{25-75\%}$ (L/s) Baseline	$FEF_{25-75\%}$ (L/s) 4 Weeks	$FEF_{25-75\%}$ (L/s) 8 Weeks
01	6.792	3.689	4.484
02	4.110	4.342	4.333
03	3.447	4.132	3.786
04	2.468	3.257	3.144
05	3.696	3.454	2.858
06	2.907	3.950	4.086
07	4.548	5.095	4.041
08	2.971	3.231	3.150
09	2.537	2.335	2.530
10	2.839	2.783	2.851
11	4.032	4.079	3.644

TABLE 4.14

FORCED EXPIRATORY FLOW FROM 25 TO 75 PERCENT VALUES FOR NCWR GROUP AT BASELINE, 4, AND 8 WEEKS

Subjects	$FEF_{25-75\%}$ (L/s) Baseline	$FEF_{25-75\%}$ (L/s) 4 Weeks	$FEF_{25-75\%}$ (L/s) 8 Weeks
01	3.076	4.507	4.219
02	2.876	2.876	4.806
03	2.521	2.805	2.828
04	4.570	3.767	3.734
05	5.054	6.640	4.862
06	3.177	N/A	N/A
07	3.716	2.622	3.703
08	3.804	3.344	2.884
09	2.679	3.026	3.944
10	3.487	4.927	3.788

4.3.4 Peak Expiratory Flow

No change in PEF in either group over the eight weeks of training was seen. However, there was an increasing trend in PEF for the CWR group (from 6.94 ± 1.20 to 7.74 ± 1.77 L/s (P value = .294)) (Table 4.15) (Figure 4.5). Individual PEF values for each group are listed in Table 4.16 and Table 4.17.

TABLE 4.15

PEAK EXPIRATORY FLOW VALUES FOR CWR AND NCWR GROUPS AT BASELINE, 4, AND 8 WEEKS

Group	PEF (L/s) Baseline	PEF (L/s) 4 Weeks	PEF (L/s) 8 Weeks
CWR (n=11)	6.94 ± 1.20	7.61 ± 1.83	7.74 ± 1.77
NCWR (n=10)	7.19 ± 2.18	7.82 ± 2.10	7.66 ± 1.11

Values are shown as mean ± SD

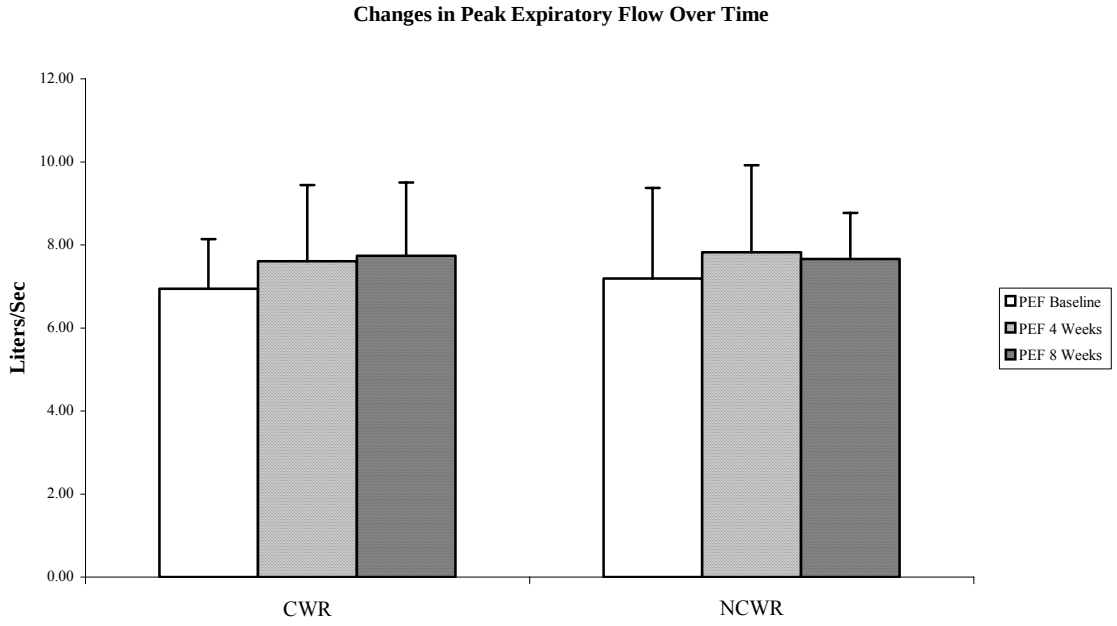

Figure 4.5. Peak expiratory flow determined during spirometry test over eight-week period.
Data are presented as mean ± SD.

TABLE 4.16

PEAK EXPIRATORY FLOW VALUES FOR CWR GROUP AT BASELINE, 4, AND 8 WEEKS

Subjects	PEF (L/s) Baseline	PEF (L/s) 4 Weeks	PEF (L/s) 8 Weeks
01	7.660	11.140	11.160
02	8.398	6.072	8.108
03	7.058	6.796	7.103
04	5.253	6.916	7.154
05	8.367	9.093	8.758
06	5.709	8.185	8.151
07	5.804	6.923	7.042
08	8.530	9.438	8.918
09	6.274	5.568	3.862
10	7.335	8.473	7.853
11	5.973	5.086	7.014

TABLE 4.17

PEAK EXPIRATORY FLOW VALUES FOR NCWR GROUP AT BASELINE, 4, AND 8 WEEKS

Subjects	PEF (L/s) Baseline	PEF (L/s) 4 Weeks	PEF (L/s) 8 Weeks
01	4.659	6.829	6.939
02	8.548	8.121	7.725
03	5.387	5.412	6.577
04	8.729	7.951	7.798
05	11.490	12.450	9.138
06	4.538	N/A	N/A
07	6.795	5.970	6.082
08	6.265	6.633	7.000
09	8.724	9.297	9.062
10	6.740	7.721	8.650

4.4 Time-to-Exhaustion

A significant difference in time-to-exhaustion was observed for both groups as participants were tested from baseline to midpoint to endpoint ($P < 0.05$). Time was recorded to the nearest 15 seconds. Follow-up post-hoc observation indicated that there was no difference in time-to-exhaustion between both groups (Figure 4.6). At eight weeks both groups had significant increases in time-to-exhaustion from 4.36 ± 2.22 to 6.11 ± 2.57 and from 4.78 ± 2.54 to 5.90 ± 2.80 minutes, respectively (Table 4.18). Time to exhaustion values for each group can be seen in Tables 4.19 and 4.20.

TABLE 4.18

TIME-TO-EXHAUSTION VALUES FOR CWR AND NCWR GROUPS AT BASELINE, 4, AND 8 WEEKS

Group	Time-to-Exhaustion (minutes) Baseline	Time-to-Exhaustion (minutes) 4 Weeks	Time-to-Exhaustion (minutes) 8 Weeks
CWR (n=11)	4.36 ± 2.22	5.59 ± 1.95	6.11 ± 2.57
NCWR (n=10)	4.78 ± 2.54	5.90 ± 2.99	5.90 ± 2.80

Values are shown as mean ± SD

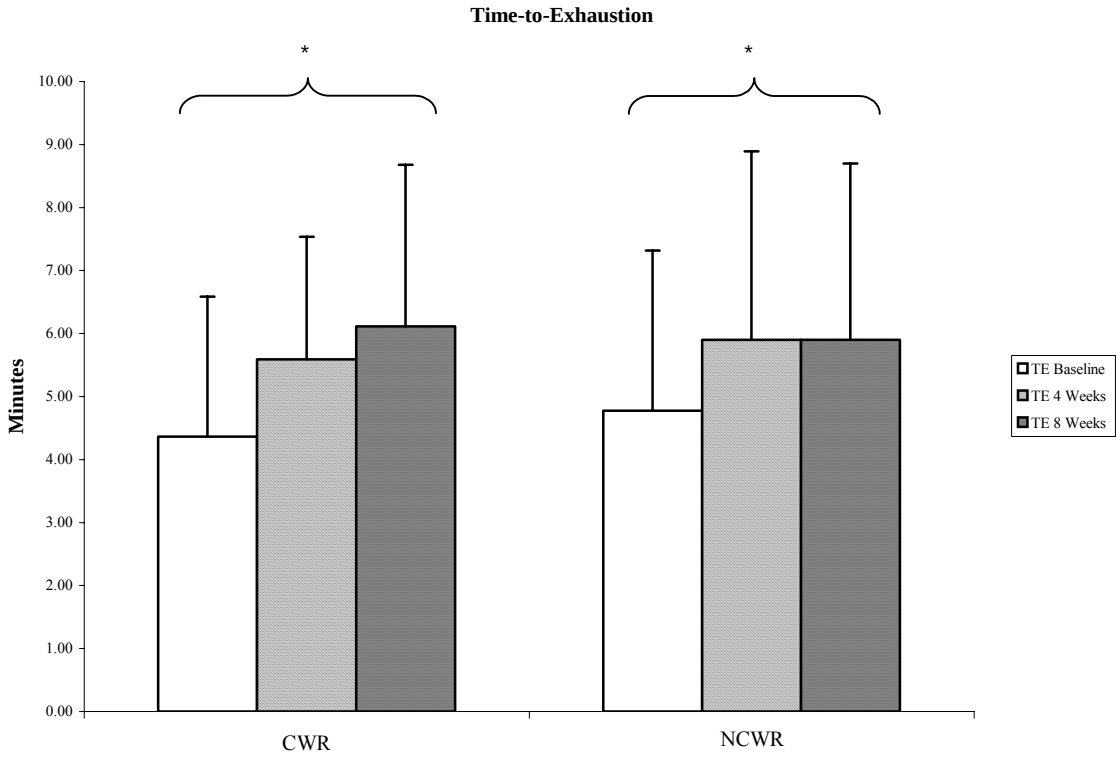

Figure 4.6. Time-to-exhaustion determined during the graded exercise test over eight-week period.
Data are presented as mean ± SD.
* $P < 0.05$ for within subject comparison
TE = Time-to-exhaustion.

TABLE 4.19

TIME-TO-EXHAUSTION VALUES FOR CWR GROUP AT BASELINE, 4, AND 8 WEEKS

Subjects	Time-to-Exhaustion (minutes) Baseline	Time-to-Exhaustion (minutes) 4 Weeks	Time-to-Exhaustion (minutes) 8 Weeks
01	4.25	4.75	5.00
02	3.00	3.25	3.75
03	4.75	4.25	4.25
04	1.75	6.25	6.25
05	5.00	6.75	7.25
06	4.25	6.50	8.00
07	3.50	4.50	5.25
08	4.75	6.50	7.25
09	2.25	4.25	5.00
10	4.25	4.25	3.00
11	10.25	10.25	12.25

TABLE 4.20

TIME TO EXHAUSTION VALUES FOR NCWR GROUP AT BASELINE, 4, AND 8 WEEKS

Subjects	Time-to-Exhaustion (minutes) Baseline	Time-to-Exhaustion (minutes) 4 Weeks	Time-to-Exhaustion (minutes) 8 Weeks
01	1.25	3.00	3.00
02	8.25	8.50	8.75
03	3.25	2.75	3.25
04	6.25	9.00	8.75
05	3.25	4.50	3.25
06	1.75	3.00	3.50
07	8.25	11.00	10.25
08	4.25	4.50	5.00
09	7.00	8.25	8.25
10	4.25	4.50	5.00

CHAPTER 5

DISCUSSION

Only eight previous studies have used chest wall restriction during moderate intensity exercise (Vanmeenen et al., 1984; Hussain and Pardy, 1985; Hussain et al., 1985; Harty et al., 1999; O'Connor et al., 2000; O'Donnell et al., 2000; Coast and Cline, 2004; Wang and Cerny, 2004). These studies showed that chest wall restriction limits individual's respiratory muscle function (Hussain and Pardy, 1985; Hussain et al., 1985; Harty et al., 1999; O'Connor et al., 2000; O'Donnell et al., 2000; Wang and Cerny, 2004), and exercise performance (Vanmeenen et al., 1984; Hussain and Pardy, 1985; Hussain et al., 1985; O'Donnell et al., 2000; Coast and Cline, 2004; Wang and Cerny). This is the first study of its kind to use chest wall restriction during exercise and show the effects of exercising with an elastic strap around the chest over an eight-week training period. This study examined whether chest restriction could be used as a training mode and the long-term effects, if any. Previous studies documented only acute responses to chest restriction during exercise.

These studies used an inelastic thoracic corset (Vanmeenen et al., 1984; Hussain and Pardy, 1985; Hussain et al., 1985; Harty et al., 1999; O'Connor et al., 2000; O'Donnell et al., 2000) and a fiberglass chest cast with inflatable cushions (Coast and Cline, 2004) to limit chest wall expansion. However, Wang and Cerny (2004) used a weighted vest that did not restrict the subject's breathing movements. The device used in this study was an elastic strap that did not prevent the chest from expanding, but offered elastic eccentric resistance for the inspiratory muscles to work against.

Previous studies used smaller numbers of participants than the 22 participants in this study: n = 12 (Harty et al., 1999), n = 5 (Hussain and Pardy, 1985), n = 5 (Hussain et al., 1985), n = 12 (O'Donnell et al., 2000), n = 11 (Vanmeenen et al., 1984), n = 18 (Coast and Cline, 2004), n = 11 (Wang and Cerny, 2004), n = 7 (O'Connor et al., 2000). The participants in this study were about the same age as those used in the other studies. Healthy, non-active individuals were recruited to participate in this study and the same population that was used in two of the eight studies (O'Connor et al., 2000; Wang and Cerny, 2004). The other studies used active individuals (Hussain and Pardy, 1985; Hussain et al., 1985) or healthy individuals (Vanmeenen et al., 1984; O'Donnell et al., 2000; Coast and Cline, 2004), thus allowing the reader to assume they were active. Of the 12 subjects used in the study by Harty et al. (1999), six subjects had previously participated in studies involving exercise testing and the level of physical fitness of the other six participants was not indicated.

5.1 VO_{2max}

In agreement with previous studies (Daniels et al., 1978; Mutton et al., 1993; Melanson et al., 1996; Sartorio et al., 2003; Egana and Donne, 2004) (Table 5.1), VO_{2max} increased significantly after eight weeks of aerobic training for both groups. However, a significant improvement of 11 ± 4 percent in aerobic capacity was observed in the experimental group at just four weeks, compared to the control group (3 ± 6 percent). These results support the hypothesis that an externally applied thoracic constriction band during aerobic exercise may assist in increasing aerobic capacity more rapidly than training without a constriction band, because of the greater increase in VO_{2max} seen within the first four weeks for the CWR group compared to the NCWR

group. For the CWR group, increases in VO_{2max} were greater between weeks one through four and started to level off through week eight. These results agree with Daniels et al. (1977), which indicated increases in VO_{2max} during the first four weeks of training but no increases after that. However, for the NCWR group, VO_{2max} continued to increase throughout the eight-week training period.

TABLE 5.1

PREVIOUS STUDIES ON VO_{2max}

Study	Length of Study (weeks)	Duration of Exercise	Test Subjects	Control Subjects	Exercise Method	Effect on VO_{2max} (percent)
Egana and Donne (2004)	12	30-40 min 3 days/wk	7	0	Running	↑ 6.6
Egana and Donne (2004)	12	30-40 min 3 days/wk	8	0	Elliptical Trainer	↑ 7.3
Egana and Donne (2004)	12	30-40 min 3 days/wk	7	0	Stair-Climber	↑ 4.8
Mutton et al. (1993)	5	4 days/wk	5	0	Cycle + Running	↑ 5.9
Sartorio et al. (2003)	3	daily for 35 min	53	0	Cycle + Treadmill + Armergometer	↑ 14.1
Iwane et al. (2000)	12	10,000 steps/day	83	0	Walking	↑ 13
Daniels et al. (1978)	8	3.2-6.4 km 5 days/wk	12	0	Running	↑ 9
Melanson et al. (1996)	9	20-40 min 3 days/wk	21	7	In-Line Skating	↑ 6
Melanson et al. (1996)	9	20-40 min 3 days/wk	17	7	Running	↑ 9.7

wk = week, min = minutes, ↑ = increase, + = combined with

A more rapid improvement was seen in the CWR group VO_{2max} within the first four weeks compared to the NCWR group. This increase in VO_{2max} may be the result of the training itself or the result of an increase in respiratory muscle strength. To determine if increases in VO_{2max} are the result of increased respiratory muscle strength, measurements of PI_{max} and PE_{max} are needed. However, studies have shown that increases in PI_{max} and PE_{max} have no direct effect on VO_{2max} (Hanel and Secher, 1991; Larson et al., 1999; Inbar et al., 2000; Williams et al., 2002)

Serious consideration must be given to the possibility that the change in VO_{2max} is not a uniquely valid index of adaptation to training. Daniels et al. (1978) suggest that although the change in VO_{2max} is a useful measure of the response to the onset of training, it is a relatively insensitive measure of the overall response to training. Future efforts to understand the training response might be directed beneficially toward other measures of the body's functioning, such as cardiac output and arteriovenous oxygen difference.

5.2 Pulmonary Function

Differences in FVC, FEV_1, $FEF_{25-75\%}$, and PEF from week one to week eight for both groups did not change. The same results were seen in previous studies after 4 to 16 weeks of IMT (Hanel and Secher, 1991; Boutellier and Piwko, 1992; Suzuki et al., 1993; Inbar et al., 2000; Covey et al., 2001; Williams et al., 2002; Gething et al., 2004). Gething et al. (2004) state that no change in FEV_1 and PEF probably demonstrates the specificity of the effects of inspiratory training on expiratory lung function, while FVC is generally dictated by the individual's stature and not influenced by training. It is in accordance with the experience gained from IMT in COPD patients as well as in normal individuals, with some exceptions (Leith and Bradley, 1976), that this type of training

does not affect standard spirometric variables (Hanel and Secher, 1991). However, pulmonary muscle function has been shown to increase after 20 to 52 weeks of aerobic training (Kaufmann and Swenson, 1981; Robinson and Kjeldgaard, 1982), contradicting the findings of this study. Duration of study may play a role in improving pulmonary muscle function; the eight weeks allowed in this study may not have been enough time to assess this adaptation.

Robinson and Kjeldgaard (1982) reported no change in lung volumes or timed VC after 20 weeks of training. However, they found significant changes in MVV, MSVC, and PIF. Kaufmann and Swenson (1981) demonstrated increases in TLC, FRC, and the FRC/TLC ratio in runner A, and similar increases in FRC and the FRC/TLC ratio in runner B. However, the measurements that displayed improvements in these two studies were not measured in this study.

5.3 Time-To-Exhaustion

Time-to-exhaustion is determined by the amount of time (in 15 second intervals) it takes the subject to reach complete exhaustion during the exercise test. Both groups demonstrated significant increases; however, there was no difference between groups, suggesting that these increases may be the result of training and not the elastic strap.

Attention has been focused on the role of the muscle tissue during habitual exercise. Increases in skeletal muscle oxidative enzyme activity with training have been well documented (Gollnick et al., 1973; Saltin et al., 1976). Reports by Henriksson and Reitman (1977) and Sjodin et al. (1976) indicated that the adaptation of skeletal muscle to training may occur independently of changes in VO_{2max}. Elite runners have been shown to have relatively high values for skeletal muscle oxidative enzyme activity

(Costill et al., 1976). These reports suggest the potential of skeletal muscle adaptation as a non-VO_{2max} related mechanism for improved running performance in response to training (Daniels et al., 1978).

In 1870, Adolph Fick developed an equation for the measurement of cardiac output in steady states of exercise (Laszlo, 2004). It states that the volume output of the heart (Q) can be calculated if the uptake of oxygen (VO_2) is measured and the amount of oxygen in each volume of arterial blood (Ca_{O2}) and mixed venous blood (Cv_{O2}) is known (5.0) as

$$Q = VO_2 / Ca_{O2} - Cv_{O2} \quad (5.0)$$

De Cort et al. (1991) investigated the relative contributions of increases in cardiac output and arteriovenous oxygen difference (A-VO_2) to the increase in oxygen consumption during exercise. As exercise increased so did Q, although most of the increase in Q was a result of increased heart rate and stroke volume. However, Q is responsible for most of the early increase in VO_2 following a sudden increase in exercise workload. They reported that after 150 seconds on a cycle ergometer, increased A-VO_2 alone would have resulted in 43 ± 7 percent of the observed increase in VO_2. De Cort et al. (1991) concluded that the early increase in VO_2 is due to increased Q. However, as exercise continues, increased A-VO_2 gradually contributes more to increased VO_2 (De Cort et al., 1991).

5.4 Inspiratory Muscle Training

Respiratory muscle training has shown to prolong constant-load exercise and cannot be explained. Earlier studies have demonstrated this phenomenon (Boutellier and Piwko, 1992; Boutellier et al., 1992). These findings weaken, to some extent, the concept

that the lung, or rather the respiratory system, is built for exercise because the fitness of the respiratory muscles is important for healthy individuals' ability to sustain constant-load exercise. Results of respiratory muscle training prolonging constant-load exercise are astonishing; the importance of the fitness of the respiratory muscles for endurance should be taken into consideration. Boutellier (1998) states that if respiratory muscle fatigue reduces performance (Martin et al., 1982; Mador and Acevedo, 1991), then increased resistance to respiratory muscle fatigue should improve performance.

Pulmonary rehabilitation is now an accepted approach for patients with moderate to severe chronic obstructive pulmonary disease. Exercise training is an important part of the rehabilitation program. After eight weeks of endurance training on a cycle ergometer, subjects with moderate to severe COPD experienced significant improvements in endurance cycling time, walking distance tests, PI_{max}, and dyspnea (Harpa et al., 2005; Pitta et al., 2004). Larson and colleagues (1999) reported that with a combination of cycle ergometer training and IMT results in significant increases in peak work rate and peak oxygen uptake, compared to individuals who only exercise or inspiratory muscle train. Studies have shown that normal endurance training fails to provide an optimal training stimulus to the inspiratory muscles and that favorable adaptation is possible with specific IMT (Romer and McConnell, 2003).

5.5 Conclusions

The following summarizes the main conclusions of this research:

1. Training with an externally applied thoracic constriction band during exercise significantly increased aerobic capacity after four weeks more rapidly than

training without the band. After eight weeks aerobic capacity was the same for both groups

2. Time-to-exhaustion significantly increased in both groups without differences between them.
3. No significant differences in pulmonary function for both groups were found.

5.6 Recommendations For Further Research

This study has raised further questions regarding the effects of chest constriction on aerobic capacity during exercise, including the potential benefit of chest restriction during exercise. The relatively short eight-week duration of this study did not provide enough statistical information to answer any of these questions. Therefore, additional research is required.

Future research might focus on respiratory muscle strength and other physiological effects, such as Q and A-VO_2, to determine what may be responsible for the improvements shown in this study. Respiratory muscle strength can be measured through the insertion of esophageal and gastric balloons to measure transdiaphragmatic pressure (Pdi) or a less invasive measurement of PI_{max} and PE_{max} using the system described by Black and Hyatt (1969). By measuring Q and A-VO_2, the researcher can determine if results are due to the type of training or other physiological effects. O_2 utilization may show improvements at the active muscle site. With additional research, specific recommendations regarding the intensity, frequency, or duration of training may be developed.

CHAPTER 6

Summary

Both groups demonstrated significant increases in VO_{2max} from week one to week eight. However, CWR showed a significant improvement (11 ± 4 percent) in aerobic capacity in the experimental group at just four weeks compared to the control group (3 ± 6 percent). No significant difference in pulmonary function was found in either group. Both groups displayed significant increases in time-to-exhaustion; however, there was no difference between the groups. This study shows that training with an externally applied thoracic constriction band during exercise significantly increases aerobic capacity more rapidly than training without the band after fours weeks.

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APPENDICES

APPENDIX A

INFORMED CONSENT DOCUMENT

EFFECTS OF CHEST WALL CONSTRICTION ON AEROBIC CAPACITY DURING EXERCISE

PURPOSE: You are invited to participate in a study to investigate whether a reduction in lung volume and chest wall movement using an externally applied thoracic constriction band over an eight-week training period, can improve an individuals' performance. The significance of this study may be that individuals can enhance their exercise tolerance more rapidly by restricting breathing and increasing their inspiratory muscle strength and endurance than those without.

PARTICIPANT SELECTION: You were selected as a possible participant in this study because your age is within the range (18-55 years) we are interested. We plan to recruit 20, healthy, non-active, adults for this study.

EXPLANATION OF PROCEDURES: If you decide to participate, you will, complete an activity questionnaire, a Physical Activity Readiness Questionnaire (Par-Q). Pre, Mid, and Post measurements will be conducted as part of the study. The pre and post testing will take place the week prior to and the week following the eight week study. Mid testing will take place in week four and will replace one day of training for that week. Only one visit is necessary for each of the testing sessions taking approximately 30 minutes to conduct the testing.

For pre/mid/post testing you will be measured for weight and height followed by a pulmonary lung function test which will measure the amount of air you can breathe out and in. Once that is completed you will perform a VO_{2max} treadmill test which requires a gradual increase in physical exertion, (i.e., the test will gradually get harder).

Following the pre testing you will be randomly placed into one of two groups: (1) Chest Wall Restriction Group (CWR), or (2) Control the Non-Chest Wall Restriction Group (N-CWR). Both groups will perform aerobic exercise three days a week for approximately 30 minutes with at least one day of rest between exercise sessions. The CWR Group will perform the exercise sessions while wearing an elastic strap around their chest. You will be encouraged to use a treadmill, elliptical machine, and/or stationary cycle randomly and in any order to complete the exercise session. Exercise intensity should be moderate at an estimated 65% to 80% of their maximal exertion. While exercising you should be slightly out of breath but still be able to hold a conversation.

DISCOMFORT/RISKS: As with any exercise program, there are some risks involved. Any time you begin new exercises, you may experience some muscle soreness. This soreness will likely subside as your body adapts to the exercises. There is also some risk of musculoskeletal injury, although you will be instructed in proper mechanics, in order

to maximize the safety of the exercise and testing, (e.g., prevent muscle strain or tear, or joint dislocation).

BENEFITS: As part of the assessment, you will receive three VO_{2peak} measurements and three pulmonary lung function tests. Participants may become more knowledgeable on how to monitor their heart rate while exercising and become more comfortable with aerobic exercise.

The information gained in this study will be particularly beneficial to exercise professionals, medical professionals, and the participants to develop appropriate training programs. This is an original study that has the potential to report an outcome that would be very beneficial to the general public and have a significant contribution to the scientific field.

CONFIDENTIALITY: Any information obtained in this study in which you can be identified will remain confidential and will be disclosed only with your permission.

COMPENSATION OR TREATMENT: Wichita State University does not provide medical treatment or other forms of reimbursement to persons injured as a result of or in connection with participation in research activities conducted by Wichita State University or its faculty, staff, or students. If you believe that you have been injured as a result of participating in the research covered by this consent form, you can contact the Office of Research Administration, Wichita State University, Wichita, KS 67260-0007, telephone (316) 978-3285.

REFUSAL/WITHDRAWAL: Participation in this study is entirely voluntary. Your decision whether or not to participate will not affect your future relations with Wichita State University. If you agree to participate in this study, you are free to withdraw from the study at any time without penalty.

CONTACT: If you have any questions about this research, you can contact me: Dr. Jeremy A. Patterson, office #112, Heskett Center, telephone (316) 978-5440. If you have questions pertaining to your rights as a research subject, or about research-related injury, you can contact the Office of Research Administration at Wichita State University, Wichita, KS 67260-0007, telephone (316) 978-3285.

You are under no obligation to participate in this study. Your signature indicates that you have read the information provided above and have voluntarily decided to participate.
You will be given a copy of this consent form to keep.

_____ _____
Signature of Subject Date

_____ _____
Witness Signature Date

APPENDIX B

PHYSICALY ACTIVITY READINESS QUESTIONNAIRE (Par-Q)

PAR-Q & YOU

Physical Activity Readiness
Questionnaire - PAR-Q (revised 2002)

(A Questionnaire for People Aged 15 to 69)
Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES NO

1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

2. Do you feel pain in your chest when you do physical activity?

3. In the past month, have you had chest pain when you were not doing physical activity?

4. Do you lose your balance because of dizziness or do you ever lose consciousness?

5. Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?

6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?

7. Do you know of any other reason why you should not do physical activity?

If you answered **YES** to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

DELAY BECOMING MUCH MORE ACTIVE:

- If you are not feeling well because of a temporary illness such as a cold or a fever – wait until you feel better; or
- If you are or may be pregnant – talk to your doctor before you start becoming more active.

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- Start becoming much more physically active – begin slowly and build up gradually. This is the safest and easiest way to go.
- Take part in a fitness appraisal – this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME_____

SIGNATURE_____

DATE_____

SIGNATURE OF PARENT_____

WITNESS_____
or GUARDIAN (for participants under the age of majority)

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.

HealthCanadaSantéCanada

APPENDIX C

EXERCISE JOURNAL

Both groups will perform aerobic exercise three days a week for approximately 30 minutes with at least one day of rest between exercise sessions. The Chest Wall Restriction Group will perform the exercise sessions while wearing an elastic strap around their chest. You will be encouraged to use a treadmill, elliptical machine, and/or stationary cycle randomly and in any order to complete the exercise session. Exercise intensity should be moderate at an estimated 65 percent to 80 percent of their maximal exertion. While exercising you should be slightly out of breath but still be able to hold a conversation. After every training period, please record what you did and for how long.

Week	Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
1							
2							
3							
4							
Testing							

Week	Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
5							
6							
7							
8							
Testing							