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THE EFFECTS OF AEROBIC AND VENTILATORY MUSCLE TRAINING ON PULMONARY FUNCTION AND SUBMAXIMAL WORK PERFORMANCE IN SUBJECTS WITH PRE-CLINICAL CHRONIC OBSTRUCTIVE PULMONARY DISEASE

A Dissertation

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Degree

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ABSTRACT

The purpose of this study was to determine the effects of aerobic training and ventilatory muscle training on pulmonary function and submaximal work performance in subjects with pre-clinical chronic obstructive pulmonary disease (COPD). Thirty-seven subjects were assigned to one of four test groups: a control, aerobic training, ventilatory training and a combination of aerobic/ventilatory training. Following a period of habituation the subjects were given pre-training tests of pulmonary function and submaximal work performance. A training period of six weeks duration followed in which the subjects' training programs were strictly monitored. Aerobic trainers rode a bicycle ergometer three times a week for a specified period of time and with a prescribed heart rate. The ventilatory trainers ventilated on a re-breathing tube five days a week for a prescribed period of time. Following the training period identical post-training tests of pulmonary function and submaximal work performance were administered. The aerobic and aerobic/ventilatory groups demonstrated an improvement in submaximal work performance with a reduction in post-training heart rate (HR) at a given workload. The ventilatory and aerobic/ventilatory groups exhibited an increase in maximum voluntary ventilation for 10 seconds (MVV_{10}) in their post-training tests. The results of this study indicate that it is possible for subjects with pre-clinical COPD to demonstrate

improvement in submaximal work performance as a result of proper training independent of changes in pulmonary function. It also demonstrates that these same subjects can show improvement in MVV_{10} with appropriate training independent of changes in submaximal work performance. Combining the aerobic bicycle training and the ventilatory muscle training did not result in a more significant increase in MVV_{10} than did the ventilatory muscle training alone, or a greater decrease in the HR during submaximal exercise than did the aerobic bicycle training alone.

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CHAPTER I

INTRODUCTION

Statement of the Problem

Many experts believe that chronic obstructive pulmonary disease (COPD) will become one of the major medical problems of the twenty first century (Bone, 1983; Higgins and Keller, 1983; Black, 1982; and MacDonnell and Segal, 1977). There are numerous factors that support this belief.

1. There has been an increase in the incidence of COPD associated with many occupations e.g., carpentry, custodial work, mining, agriculture. These workers continually breathe air contaminated with minute particles of debris (Andersen et al., 1979).
2. This century has seen a great population increase in the urban areas. In many of these areas where air pollution has constituted a serious health hazard, there has been a corresponding increase in the number of patients who suffer from COPD (Mitchell, 1978).
3. Smoking is part of a lifestyle of a significant number of people and there is an extremely high incidence of COPD in the smoking population (Mitchell, 1978; Tattersall, et al., 1978).
4. There have been many advances in the medical field relating to the treatment of COPD, including improvement in drug

> and antibiotic therapy. In the past, the majority of the people who began to demonstrate the clinical signs and symptoms of COPD died of respiratory failure usually associated with pneumonia. Advances in medical technology have prolonged the lives of these individuals and as a result, we are now observing the crippling effects of COPD in its advanced stages (Hale et al., 1978).

A major problem with this particular disease process is that it is insidious in nature and in many cases, the victims are not aware of their medical problem until the disease has reached an advanced stage (MacDonnell and Segal, 1977; Hale et al., 1978). The reason for this is that normal sedentary individuals use approximately 20% of their respiratory system's reserve capacity during their normal activities. As a result, the early compromise of the respiratory system goes unnoticed. Early clinical symptoms usually include chronic cough and shortness of breath with exertion. By the time the subject is aware of any respiratory disorder, the disease may have already progressed to an advanced stage (Hale et al., 1978; Petty and Cherniak, 1981).

In the past two decades, numerous studies have been conducted regarding the effects of aerobic and ventilatory exercises on individuals in the advanced stages of respiratory disease (see reviews by Belman and Mittman, 1980; and Hale et al., 1978). Some researchers have demonstrated that patients in the advanced stages of COPD can improve their work tolerance following particular

training programs. This increase in work tolerance is normally demonstrated on such devices as a treadmill or bicycle ergometer. To date, however, it is generally acknowledged that although work tolerance may be improved, there appears to be no alteration in the pulmonary function of these individuals (Alison et al., 1981; Belman and Kendregan, 1982; Hale et al., 1978). In the last few years, the medical profession has become increasingly aware of its inability to treat COPD in its advanced form and has begun to shift its emphasis to the prevention of this disorder through early detection (Black, 1982).

Purpose of the Study

The purpose of this study was to determine the extent to which specific pulmonary function and physical work capacity measures associated with COPD could be improved through aerobic training, ventilatory training, or a combination of aerobic and ventilatory training.

Limitations of the Study

1. Subjects who exhibited early diagnostic signs of COPD without manifesting clinical symptoms, and who, under normal circumstances would not be expected to seek medical attention for their disease process for many years, were selected. These subjects ranged in age from 21 to 45 years. These individuals were selected for this study because the percentage of their observed forced expiratory

volume for one second (FEV_1) divided by their observed forced vital capacity (FVC) was less than the predicted value for normals of the same sex, age, and height (Hewlett-Packard's Pulmonary Function Vital Capacity Manual, HP/67/69, User's Library Program).

2. Only 37 subjects took part in this study. For the most part, these individuals were employees of Copley Hospital in Morrisville, Vermont or members of employees' families. Although expected or desired results were not explained to these subjects, many of them anticipated the expected results by virtue of their training and experience.
3. The training program lasted six weeks, and the post-training tests were administered not less than three days after the individual's last training period. The results of this study reflect the changes immediately following their training program and should not be used to predict long term changes.
4. Participants in this study, for the most part, showed borderline or mild signs of COPD (resistance to air flow) without any clinical manifestations. We cannot assume that individuals with severe COPD would respond in the same manner to this type of training procedure.
5. Only the submaximal work tolerance and ventilatory aspects of COPD were evaluated in this study. There is no mention made of changes in gas diffusion or alterations in the blood gas concentrations.

CHAPTER II

REVIEW OF LITERATURE

Introduction

The pertinent review of literature may be classified under nine major categories. They are:

1. Mechanics of Respiration,
2. Anatomical and Physiological Changes Associated with Chronic Obstructive Pulmonary Disease,
3. Clinical Manifestations of COPD,
4. Diagnostic of COPD,
5. Aerobic Training in Normal Individuals,
6. Aerobic Training in Patients with COPD,
7. Respiration as a Limiting Factor in Physical Work Capacity,
8. Ventilatory Muscle Training in Normal Subjects,
9. Ventilatory Muscle Training in COPD Patients.

Mechanics of Respiration

The thoracic cavity is that region between the abdomen and the neck. It is the largest of the body cavities, whose bony landmarks consist of the twelve thoracic vertebrae, their ribs, and the sternum. Its inferior boundary is the muscular diaphragm. The thoracic cavity is divided into three parts. The first is the mediastinum which contains the heart and its great vessels, the

trachea, and esophagus. The second two compartments are the right and left pleural cavities. The pleural cavities are created by thin-walled sacs of serous epithelium which envelope the lungs (Basmajian, 1971). The pleural lining itself has three components. The outer component is the parietal pleura which adheres to the internal surface of the thoracic cavity. The inner layer is the visceral pleura which invests the lungs. The intermediate area is known as the isthmus which is filled with a serous fluid and creates a potential cavity with a negative pressure. This lubricated space allows the two membranes, parietal and visceral pleura, to slide back and forth on one another, yet at the same time they remain close together when the volume of the thoracic cavity changes in respiration (Guyton, 1976; Basmajian, 1971).

The muscles of respiration attach to the bony thorax and are comprised of four basic groups: the diaphragm, the intercostals, abdominals and other accessory muscles. The primary muscle of respiration is the diaphragm. In its relaxed state it is a dome-shaped muscle with its convex surface moving superiorly. In a contracted state, the dome-shape is diminished and presses down on the abdominal viscera increasing the length and volume of the thoracic cavity. The accessory muscles (scalene, sternocleidomastoid, and trapezius) and the external intercoastals become active with increased ventilatory activity. Their functions are to elevate and/or fix the ribs, increasing the anterior/posterior diameter of the thorax. The abdominals and the internal intercoastals function mainly as

expiratory muscles. During quiet ventilation, exhalation is produced by the passive recoil of the lungs. However, when increased ventilatory demands are made, passive expiration does not occur rapidly enough and the expiratory muscles are utilized. Their major function is to pull the ribs inferiorly and medially (Luce and Culver, 1982).

Functionally the lungs consist of two types of air spaces. The first type is comprised of conducting airways which move bulk gas in and out of the lungs. The second type is made up of the small air spaces where gases are exchanged with the pulmonary blood. Anatomically these two major zones of the lungs are not clearly separated because of a transitional structure, the respiratory bronchioles, which function in both capacities (Ryan, 1981). Spearman and Sheldon (1982) described 23 generations of branches as the airways subdivide from the periphery of the lung to the alveolus. They stated that the first 16 generations roughly constitute the conducting zones; the seventeenth through the nineteenth branches were the transitional zone of the respiratory bronchioles; and the twentieth through the twenty-third, the terminal alveolar ducts, are the site where the main diffusion occurs.

The trachea divides into the right and left main stem bronchi and then into small bronchi. These structures are reinforced by cartilaginous plates. As the diameter of the bronchi becomes smaller, the cartilaginous plates become less frequent. The internal surface of these structures are lined with ciliated cells. "The many hair-like cilia on the surface of each cell beat rhythmically to propel a

blanket of mucus upwards toward the larynx" (Ryan, 1981). As the diameter of the airways continue to reduce and approach 1 to 2 mm, the cartilage plates cease and the structures become known as the bronchioles. Many mucus glands are located in all the structures from the trachea to the bronchioles and function to moisten the airway, trap particulate material and prevent irritation (Guyton, 1976; Ryan, 1981). The walls of the bronchi and bronchioles contain an abundant amount of smooth muscles which are arranged circumferentially so that contraction will cause a narrowing of the airways, and relaxation will cause a broadening of the airways. The smallest bronchioles are known as respiratory bronchioles and are transitional in nature. Their major responsibility is the transportation of bulk gas, but some diffusion with pulmonary blood occurs across their surface. The respiratory bronchioles give rise to the alveolar ducts and the terminal alveoli. It is in this area that the bulk of the diffusion between the blood and the lungs occurs (Ryan, 1981). While the larger airways are maintained by their cartilaginous skeleton, the smaller, delicate air spaces are maintained by a series of connective tissue (collogan and elastic fibers). These very small airways consisting of the respiratory bronchioles, alveolar ducts, and alveoli are also bounded by a network of very fine pulmonary capillaries.

According to many writers (e.g., Ryan, 1981; Guyton, 1976), the work of respiration occurs mainly during inspiration and comes from three different sources: the first is the elastic resistance

of the respiratory system; the second is the tissue viscosity itself; and third, is the resistance to the movement of air through the air passages. This airway resistance increases with the velocity of the ventilation. At any given velocity, the resistance to air flow may be reduced by bronchial dilation or increased by bronchial spasm. the movement of air in or out of the lungs is referred to as pulmonary ventilation. At rest, inspiration is mainly a function of the contraction of the diaphragm while expiration is the passive recoil of the pulmonary tissue by virtue of its collogen and elastic fibers. During work or exercise, both inspiration and expiration are produced by active muscle contractions and the diaphragm is assisted by the actions of the muscles of the abdomen, intercostals, and accessory muscles (Luce and Culver, 1982).

Anatomical and Physiological Changes Associated With COPD

Chronic obstructive pulmonary disease is not a specific disease process, but rather a grouping of specific diseases with similar clinical manifestations (Ryan, 1981; Burton et al., 1977; MacDonnell and Segal, 1977). The clinical sign which is common to all aspects of COPD is the "chronic unremitting obstruction to flow of air in the airways of the lungs" (Ryan, 1981).

Regardless of the specific disease process, the abnormal resistance to air flow occurs in one of three places; luminal obstruction, airway wall narrowing or extramural wall occlusion

(Hoidal and Niewoehner, 1983; Ryan, 1981; Burton et al., 1977; MacDonald, 1980).

1. Luminal obstruction is associated with excessive secretions which clog the airway. These excessive secretions are the result of the increased mucus production secondary to chronic irritation or infection.
2. Airway wall narrowing is caused by smooth muscle constriction, hypertrophy of the submucosal glands, swelling of the basement membrane, and an increase in the presence of scar tissue.
3. Extramural wall occlusion is associated with the destruction of the supporting parenchymal structures, (collagen and elastic fibers) which support the alveoli, and chronic pulmonary edema.

COPD in its various forms is quite common today. It is estimated that between 5% and 20% of the adult male American population has COPD in some form or another (MacDonnell and Segal, 1977; Black, 1982). The two most common types of COPD are emphysema and bronchitis. It has been found that in severe cases of COPD, it is usually a combination of both these disorders (Mitchell, 1978).

In chronic bronchitis there are lesions associated with the bronchi and smaller bronchioles. Bronchitis is clinically defined as a cough with sputum that lasts three months out of a year for at least two consecutive years. In addition, there is the proliferation of the mucus glands in these bronchi and bronchioles (Mitchell, 1978;

Burton et al., 1977). Over time, chronic bronchitis becomes chronic obstructive bronchitis with morphological changes in the bronchi and bronchioles, including an increase in size and number of the submucous glands, excessive mucus production, increased thickness of the basement membrane with scar tissue present, constriction of the bronchiole smooth muscles and on the epithelial surface, there is denuding of the cilia in a random fashion. These so-called "skip areas," are areas where sputum can only be propelled by coughing or suction (Ryan, 1981; Burton et al., 1977). Chronic bronchitis therefore obstructs air flow by compromising the lumen with excessive secretions and by causing hypertrophy of the walls of the bronchial airways due to chronic inflammation and scarring.

Emphysema is the second major form of COPD and is characterized by the enlargement and eventually the destruction of the terminal airways. Anatomically, emphysema can be categorized as in centrilobular emphysema where the lesion is found in the respiratory bronchioles and panlobular emphysema where the lesion is found in the alveoli themselves. In either case with the initial enlargement of these terminal airways, a diffusion problem exists because of the decrease in membrane surface per unit volume of air (Burton et al., 1977). The continued progress of this disease finds the destruction of the extraluminal structures and the loss of radial traction which maintains the internal lumen of these small airways. Destruction of the collagen and elastic fibers allows many of these airways to collapse and reduces the elastic recoil that is

associated with the healthy lung (Ryan, 1981). According to Ryan and others, (e.g., MacDonnell and Segal, 1977), chronic bronchitis and emphysema will often co-exist causing airway obstruction by:

1. Retaining mucus which tends to plug the small airways;
2. Chronic inflammation with scarring and narrowing;
3. Patients with COPD are subject to repeated episodes of acute infections, and the inflammatory exudate associated with these infections can exacerbate the airway obstruction; and
4. Loss of radial traction caused by emphysema causes obstruction in those small airways (Ryan, 1981).

Bronchial asthma which is the hyper-reactive state of the smooth muscle in the bronchioles themselves, has been classified as a clinical syndrome associated with COPD by the National Tuberculosis and Respiratory Disease Association in 1972 and authors since that time (Burton et al., 1977). Clinical findings change markedly during periods of time when bronchial spasm exists, causing an increased resistance to air flow. In this particular study, asthmatic patients were excluded because of the extreme changes in their pulmonary function between times of exacerbation and remission of their disease process.

Clinical Manifestations of COPD

In the past, the prognosis for COPD patients has been grave. They have been condemned to a recurring cycle of hospitalization,

inactivity at home, recurrent infection, and rehospitalization (May and Kenny, 1980). According to Hale et al. (1978), a major reason for this morbidity is that medical care is not sought until the disease has passed into an advanced stage, when the individual's cardiopulmonary reserve falls below their physiological demands. This leads to acute episodes of dyspnea with activity and the individual's fear of exertion which decreases the individual's level of activity and leads to further cardiopulmonary deterioration.

The irritated pulmonary tissue of a COPD patient causes continued excessive mucus production which decreases the efficiency of the respiratory system, and increases the incidence of pneumonia (Petty and Cherniak, 1981). With today's advances in medical treatment, these individuals are able to survive these recurring episodes of acute respiratory distress. However, the chronic irritation and recurring infections cause scarring and plugging of the airway, effectively reducing the size of the lumen. This decrease in the luminal radius increases the resistance to air flow, which increases the work of ventilation. As a result, the patient begins to suffer dyspnea with exertion. If the disease process continues, destruction of the bronchiole and alveolar walls begins. In time, this leads to the vasoconstriction of the pulmonary capillaries to the under-ventilated alveoli causing pulmonary hypertension (Nett, 1982). In the presence of pulmonary hypertension, the right ventricle muscle pumps against increased pressures leading to ventricular hypertrophy. The right ventricle cannot compensate for the increased pulmonary

pressure indefinitely, and right side heart failure (cor-pulmonale) begins. "All COPD patients will get cor-pulmonale if they live long enough" (Meador, 1980).

The magnitude of the problem is great. COPD is second to heart disease as the leading cause of death in this country (Ryan, 1981). A report by the Committee on Thoracic Medicine to the Royal College of Physicians (1981) stated that in the 15 year study, 10% occupancy of the medical beds in hospitals in England were due to COPD. The cost in pounds and the lost man hours from work were staggering.

In recent years, however, aggressive rehabilitation efforts have been made on behalf of these patients with some modest success. The treatment program includes prophylaxis, cessation of cigarette smoking, the removal of occupational pollutants, drug therapies, antibiotics for acute infections and bronchial dilators for dyspnea, exercise training, and the home oxygen therapy (Harrison, 1982).

Diagnosis of COPD

One of the early methods for diagnosing the presence of COPD was through radiological abnormalities which appeared on patients who were continually being treated for respiratory ailments. In order to make a diagnosis of COPD through radiographic interpretation, abnormalities in one or more of four general areas must be present. The first diagnostic clue to the possibility of COPD is the chronic over-inflation of the lungs. This is determined by the flattened

appearance of the diaphragm during expiration with the superior portion near the area of the seventh rib or lower (McCarthy et al., 1975). Furthermore, there appears to be a reduction of the excursion of the diaphragm between maximum inhalation and maximum exhalation. A second indication of the presence of COPD is the early narrowing of the pulmonary vessels as they enter the lungs. In the presence of COPD, localized zones of narrowing appear in these vessels and are associated with the condition of cor-polmonale or pulmonary hypertension (Nicklaus et al., 1966). A third area indicative of COPD is evidence of alveolar destruction.

> The only direct evidence of alveolar destruction is a hyper-transradiant area without vessel shadows within it. Alveolar loss is presumed to be present if in other parts of the lung there are vessels which are normal or even dilated which can be used as marker vessels (Simon, 1956).

The fourth area in which radiological clues to the presence of COPD can be made, is the evidence of an increase in the retrosternal and retrocardiac spaces, evaluated on a lateral view of the thorax (L. Ellenbogen, M.D., personal communication, January 20, 1984). Although radiologic analysis can lead to the definitive diagnosis of COPD, radiographic proof can be found only in the advanced stages of the disease process. As a result, x-ray analysis as a screening procedure for early COPD is not appropriate. To date, the most common method for determining the presence of early COPD is through basic pulmonary function analysis (L. Ellenbogen, M.D., personal communication, January 20, 1984; Belen, 1977; Epler et al., 1980; Black, 1982).

Pulmonary function measurements have been used for the determination of COPD for a long period of time. The importance of the early diagnosis of COPD for effective treatment is of paramount importance (Bruderman, 1976). As a result, there is continued effort being made to develop new methods for the early detection of this disorder. One method is the analysis of the "closing volume." The closing volume is representative of that air which is trapped in the lungs in excess of the residual volume as a result of the collapse of the small airways. There are numerous methods to determine closing volume. One of the most common used is the single breath Nitrogen Washout Procedure. In this procedure, the patient sits quietly, breathing room air. After a few minutes the patient is instructed to forcefully exhale completely. This rapid forceful exhalation causes some of the smaller damaged airways to collapse trapping air. Inhalation then occurs using the gas of 100% oxygen. Following the inhalation, the patient immediately exhales slowly into a spirometer which measures the Nitrogen content and the expired volume. Initially the expired gas contains 100% oxygen, as it empties from the larger airways. As exhalation continues to occur in a slow manner, the smaller airways which collapsed during forced expiration trapping the air, begin to empty. At this time, Nitrogen appears in the spirometer. This is normally reported as the ratio between the closing volume and the vital capacity (Bruderman, 1976; Spearman and Sheldon, 1982). The larger the percentage of Nitrogen, the greater the air trapped, and the more severe the COPD.

Another new method which is beginning to be used is the method known as the "Flow Volume Loops." This utilizes an electronic spirometer in which the maximum forced expiratory volume is followed immediately by a maximum inspiratory flow volume. Clinically the initial portion of the maximum expiratory volume is effort dependent. However after the initial expiratory effort, the remaining flow volume is effort independent and reproducible. Reduced flow at this point on the flow volume loop is indicative of small airway obstruction. The inspiratory portion of the flow volume loop is more sensitive to central airway obstruction which is indicative of the disease process in the larger airways (Bruderman, 1976; Spearman and Sheldon, 1982).

Ashutosh and Keighley (1978) suggest that the most sensitive test for early detection of COPD is the evaluation of air flow during passive expiration. It was felt that the free collapse of the lungs following maximum inhalation was solely based on the force of the elastic recoil of the tissues, rather than by the voluntary effort of the skeletal expiratory muscles. They used this technique to evaluate the difference between smokers and non-smokers who had similar scores on their forced vital capacity (FVC's), forced expiratory volume for one second (FEV_1's), and forced expiratory flow between 25-75% of vital capacity ($FEF_{25/75\%}$). The results showed a significant difference between the smokers and non-smokers with similar pulmonary function tests, leading these investigators to theorize that this is the most sensitive test for small airway obstruction.

Continued research is going on and is necessary to determine more sensitive and reliable indicators of early COPD. At the present time, however, the standard by which a person is assessed for the presence or absence and the severity of COPD is through the use of basic spirometry. The pulmonary function tests most commonly used are FEV_1 and FVC (Black, 1982; Solomon, 1978; Spearman and Sheldon, 1982; Ryan, 1981; Kory et al., 1961). Epler et al. (1980) selected FVC and FEV_1 as screening tests because, "They are required for most published schemes, they are performed routinely in most clinical laboratories, and they are mandatory for epidemiologic studies."

Aerobic Training in Normal Individuals

Aerobic activities increase the oxygen consumption of individuals. This occurs when large muscle masses are used in rhythmic contractions over a period of time. Improvement in aerobic performance depends on adaptations of the cardiovascular system and the skeletal muscles (Froelicher, 1973; Ekblom et al., 1968; Saltin, 1969; Balke and Ware, 1959). These adaptations can be evaluated as acute changes (physiological changes that occur from rest to exercise) or chronic adaptations (changes in the individual over a prolonged period of time) (Wilmore, 1977). The major function of the cardiovascular system during activities is the transportation of O_2 and nutrients to active tissue and the removal of metabolic waste products. The acute cardiovascular adaptation or response from rest to exercise include an increase in heart rate and stroke

volume to provide a greater cardiac output, a redistribution of blood from non-active to active tissue, a concomitant increase in blood pressure to assure adequate perfusion of these tissues and an increase in oxygen extraction from the blood creating a greater arterial venous oxygen difference (a-v O_2 diff.) (Rowell, 1974; Astrand and Rodahl, 1977; Wilmore, 1977). These changes summarize the acute adaptations of the cardiovascular system to exercise.

Chronic adaptations of the cardiovascular system to habitual exercise can be identified in individuals. There is a reduction in heart rate of 10 to 30 beats/min. at rest and at a given sub-maximal workload as the result of aerobic training (Ekblom et al., 1968; Rowell, 1974). There are no increases in maximal obtainable heart rates with training. A rapid return to resting heart rate following exercise is also reported with chronic training. An individual's stroke volume may increase 10 to 20 ml/beat at rest and 20 to 30 ml/beat during submaximal exercise (Rowell, 1974; Wilmore, 1977). Rowell (1974) reports that this increase in stroke volume is found only in young and middle-aged subjects. Older subjects increase their cardiac output by heart rate alone.

Ekblom et al. (1968) evaluated cardiac output during maximal exercise to compare sedentary and trained individuals, as well as championship endurance athletes. The range of cardiac output for sedentary individuals was approximately 20 to 25 liters per minute; for trained individuals 25 to 30 liters per minute, but with championship athletes maximum values exceeded 40 liters per minute.

Interestingly, there was no difference between the groups in maximum obtainable heart rate. The significant difference was found in stroke volume with sedentary individuals demonstrating maximum stroke volume between 100 and 120 ml/beat; trained individuals 130 to 155 ml/beat; and championship athletes 175 to 200 ml/beat. This difference between sedentary and trained individuals is probably a result of cardiac hypertrophy and an increased ventricular volume associated with training. The excessive stroke volume of successful athletes may be genetic, as well as training induced (Wilmore, 1977).

Chronic changes in a-v O_2 diff. are somewhat controversial, but may increase as a result of training through better redistribution of blood volume and increased vascularization of working tissues (Saltin, 1969; Wilmore, 1977). The VO_2 at rest and during submaximal work is unchanged with training, but with maximal exercise there is an increase in VO_2 max of about 15% to 30% in normal individuals (Wilmore, 1977).

Stoedefalke (1977) reviewed exercise studies in middle-aged males and reported modest improvement in aerobic power and fitness following 20 to 30 weeks of regular exercise if training thresholds are reached which include a frequency of exercise 3 to 4 times a week at an intensity of 70% of the individual's maximum aerobic capacity and the duration of 20 to 45 minutes.

Aerobic Training in Patients with COPD

The fact that exercise intolerance is a clinical manifestation of COPD is generally accepted. The mechanisms of this intolerance

were reviewed by Brown et al. (1977) and include the following:

1. Ventilatory inefficiency caused by an increase in the anatomical dead space in comparison with the tidal volume (V_D/V_T).
2. Gas exchange abnormalities caused by the alteration or destruction of the respiratory membranes and/or the pulmonary capillaries.
3. Obstruction to air flow which increases the work of breathing and limits maximal exercise ventilation.

In his review, Shephard (1977) identified invalidism as another possible mechanism. Bed rest can reduce the maximal oxygen consumption of an individual to 30% below that of the predicted value for sedentary normals. In COPD patients, dyspnea and fear of exertion limit activity and cause a continuing cycle of de-conditioning.

Pierce et al. (1964) examined exercise training in nine subjects with severe COPD. Pre-training pulmonary function and ventilatory capacities were evaluated, as well as heart rate, respiratory rate, minute volume, VO_2 and VCO_2 during a 2.5 minute treadmill walk at the subject's maximum obtainable speed. Training consisted of 2.5 minute walks on a treadmill slightly below the subject's maximum attainable speed 5 to 10 times a day. Between each exercise bout, the subjects' vital signs returned to their resting values. The training program for these subjects lasted from 2 to 20 weeks. Using this interval type training, the

investigators reported an increase in work tolerance demonstrated by a decrease in heart rate, respiratory rate, minute volume and $\dot{V}O_2$ at a given submaximal load. They also reported increased work capacities because the subjects were able to obtain higher speeds on the treadmill. The investigators reported, however, that there were no changes in pulmonary function or ventilatory capacities.

Nicholas et al. (1970) evaluated eight patients with COPD in a three month control period followed by a six month exercise therapy period with exercise administered three times a week. These exercise sessions consisted of three 10 minute walks at increasing intensity. In addition, the subjects were given a set of Master's two steps and told to practice daily at home for 10 minutes. The patients were also encouraged to walk as much as possible. No significant changes occurred in the pulmonary function tests or in arterial blood gases; however, four of the eight demonstrated considerable gains in exercise tolerance. The authors theorized that the improvement of work tolerance in the absence of improving pulmonary functions is probably due to the loss of fear of exertion and dyspnea, and increased muscle strength and efficiency.

In 1978, Mertens et al. examined the effects of exercise on COPD patients over a prolonged period of time. Thirteen subjects with mild to moderate COPD who demonstrated exercise intolerance and led sedentary lives were selected. Following the initial evaluation, individualized graded walking and jogging programs were established. Each subject had one supervised exercise period per

week and then participated at home for the remainder of the days. This group of investigators used several pre- and post-training fitness appraisals such as body build, muscle strength, muscle endurance, and resting heart rate. Progressive exercise bicycle tests were used in which heart rate, respiratory rate, and minute volume were evaluated. Various lung functions were also included: FVC, FEV_1, functional residual capacity, and diffusion capacity. These investigators found that this type of endurance program had little effect on body build, muscle strength, or lung functions. However, there were gains in quadricep endurance, a decrease in resting heart rate, and a decrease in heart rate with graded exercise. Their study confirmed what previous studies had reported: there can be substantial symptomatic improvement in COPD patients with exercise training without associated improvement in lung function.

Mungall and Hainsworth (1979) evaluated 10 COPD patients over three 12-week periods. The first period served as a control. The second period consisted of daily exercises, 12-minute physical fitness programs from the Royal Canadian Air Force Program, 5BX/XBX (1964). During the third period, the subjects returned to their normal sedentary living. These investigators reported an improvement in the 12-minute walk after the training period and following the period of de-training. However, they felt that the improvement that was sustained during the period following training may have been caused by virtue of the fact that many of the subjects continued

to exercise rather than return to their sedentary lifestyle. These investigators reported small but significant increases in FEV_1 immediately following the training period. It should be pointed out, however, that measurements of FEV_1 were made at 2-week intervals during all three of the phases of this program, and it may be possible that some learning occurred.

Alison et al. (1981) evaluated the effects of a 12-week progressive bicycle program on the distance COPD patients walked in 12 minutes and FEV_1. There was a significant increase in the distance walked in 12 minutes without significant change in FEV_1. Brown and Wasserman (1981) reviewed the effects of exercise on COPD subjects and stated that even though pulmonary functions did not change, this type of exercise was beneficial due to the increase in work tolerance and an improvement of the quality of life of these individuals.

Respiratory Factors and Their Possible Role in Limiting Physical Work Capacity

Traditionally, the respiratory system has not been thought to limit exercise in normal individuals (Rowell, 1974). However, much experimentation will be required to prove or disprove this theory. In a review, Bye et al. (1983) examined the role of respiratory factors as possible limiting factors in exercise. According to the authors there are four possible areas where respiration may, in fact, limit physical work capacity. These areas are:

1. Pulmonary Gas Exchange;
2. Mechanics of Air Flow;
3. Energetics; and
4. Respiratory Muscle Fatigue.

Pulmonary gas exchange can be evaluated by the arterial oxygen pressure (PaO_2) or by the alveolar-arterial oxygen gradient ($AaDO_2$). The fact that hypoxia causes reduced physical work capacity in normal subjects has been well documented at high altitudes. Hypoxia has been discovered in some superior athletes working at high intensities and in patients with a variety of lung diseases (Bye et al., 1983). However, the bulk of the evidence collected on athletes and sedentary normals show that the resting PaO_2 is maintained even in exhaustive exercises (Rowell, 1974). According to Dempsey et al. (1979; 1982), in those cases where highly trained and motivated athletes were able to produce exercise hypoxemia, the condition was corrected when the athletes breathed a mixture of 80% helium and 20% oxygen. The breating of this low density gas returned the PaO_2 to its normal value. Dempsey theorizes that the respiratory system could have corrected the hypoxemia if the ventilation of more dense atmospheric air had increased. In the case of patients with lung disease a ventilation perfusion mismatch often occurs, causing exercise hypoxia. In emphysema, this is due to the destruction of the alveolar wall, elastic tissues, and surrounding capillaries (Ryan, 1981; Burton et al., 1977); in bronchitis, it is due to the inability to adequately ventilate the alveoli caused by narrowing of the airways (Mitchell, 1978).

Bye et al. (1983) theorized that the increase in ventilation with HeO_2 by the athletes who demonstrated hypoxemia while breathing air, indicated that other factors, not diffusion, may limit exercise tolerance, particularly the mechanics of ventilation, muscle fatigue, or the energetics of respiratory muscles.

The mechanics of air flow has been shown to limit exercise in normal subjects by having them breathe dense gases which increase the work of respiration (Wood and Bryan, 1978). Here the individuals were forced to terminate their exercise at levels of oxygen consumption and heart rates which were well below their normal maximum values. In subjects with COPD, the increased work of respiration caused by the resistance to air flow is similar to that experienced by the normal individuals breathing dense gas. It has been well documented that patients with COPD are forced to terminate their exercise at levels of oxygen consumption and heart rate that are far below their predicted values expected for individuals of similar age and sex (Leaver and Pride, 1971; Stubbing et al., 1980). Another example of how mechanics of air flow may limit exercise performance in patients with COPD is that their exercise ventilation equals their maximum breathing capacity (Clark et al., 1969; Potter et al., 1971). The same is not true with normal subjects (Tenney and Reese, 1968; Shephard, 1967).

A measure of the energetics of respiration is the O_2 cost of breathing. At rest, the oxygen cost of respiration is considered to be minimal (Otis, 1964). During maximal exercise, there appears

to be a substantial increase in this value. Furthermore, Tenney and Reese (1968) suggest that at high ventilations (in excess of 120ℓ/minute), the oxygen cost of ventilation increases disproportionately to the increase in ventilation, and becomes ineffective in providing more oxygen to the working tissue. Otis (1964) suggested that a level of ventilation could be reached where any further increase in VO_2 would be consumed entirely by the respiratory muscles. He estimated this critical value to be approximately 140ℓ/minute. The patient with COPD faces a greater disadvantage, and the pathological changes of the disease process increases the resistance to air flow which requires greater respiratory effort to produce the same pulmonary ventilation as normals (Ryan, 1981).

Respiratory muscle fatigue, as defined by Bye et al. (1983), is the "inability of the respiratory muscles to continue generating a given pressure." It has been demonstrated that respiratory muscle fatigue does occur during ventilatory tasks. Roussos et al. (1979) demonstrated respiratory muscle fatigue when ventilation occurred at pressures greater than 40% of the subject's maximum inspiratory pressure. Furthermore, respiratory muscles have limited endurance in performing ventilations that approach 80% of their maximum breathing capacity over a prolonged period of time (Freedman, 1966, 1970; Tenney and Reese, 1968). However, the question of whether or not respiratory muscle fatigue does occur as a result of regular exercise is still unclear. In the case of patients with COPD, however, for a given VO_2, they are required to

ventilate against an increased resistance by virtue of the resistance to air flow. In severe cases, COPD patients have a greater pulmonary ventilation for a given VO_2 due to the destruction of the diffusion membrane which exists in the respiratory bronchials and alveoli.

In summary, the question of whether or not the respiratory system limits exercise performance in normal subjects is still one of controversy and requires further research; however, the speculation that respiratory function does limit work tolerance in patients with COPD appears evident due to their increased resistance to air flow and destruction of the diffusion membranes.

Ventilatory Muscle Training in Normal Subjects

To date there has been limited work done on the specific effects of ventilatory training on normal subjects. Leith and Bradley (1976) evaluated a five week training program of the ventilatory muscles on normal subjects. Prior to training, the vital capacity, total lung capacity, maximum voluntary ventilation for 15 seconds (MVV_{15}), maximum sustained ventilatory capacity for 15 minutes (MSVC), and maximum inspiratory and expiratory pressures were evaluated. The training groups consisted of control, strength trainers, and endurance trainers. The five week training program was held five days a week for a period of 30 to 45 minutes per day. The strength trainers performed repeated static maximum inspiratory and expiratory maneuvers against obstructed airways while the endurance trainers performed voluntary normocapnic hyperpnea to exhaustion.

The results of the five week training program showed a 55% increase in maximum pressures of the strength trainers and an increase in vital capacity and total lung capacity of approximately 4%. The endurance trainers increased their MVV_{15} by 14%, and were able to sustain 96% of their MSVC for 15 minutes. Prior to training, they were able to sustain only 81% of their MSVC. In 1977, Keens et al. demonstrated a similar phenomenon with endurance training using four normal subjects as a control group. These individuals trained five days a week for four weeks at sustained maximal normocapnic hyperpnea. Following the four week training period, these subjects demonstrated a 22.1% increase in their MSVC over a 15 minute period.

In 1980, Haas and Haas investigated the effects of strength training of inspiratory muscles on various pulmonary functions and on maximum O_2 consumption (VO_2 max) attained on a graded exercise treadmill test. The training program was approximately three weeks, during which time the subjects attempted to maintain 85% of their maximum inspiratory pressure until fatigue occurred. The post training results demonstrated an increase in strength of the respiratory muscles, as well as an increase in maximum voluntary ventilation. There was, however, no change in the VO_2 max on a graded treadmill test.

In 1981, Morgan (unpublished thesis, The University of Tennessee, Knoxville) attempted to determine the effects of respiratory muscle endurance training on maximal oxygen uptake. The

subjects were evaluated in a 15 second maximum voluntary ventilation (MVV_{15}) test and an endurance breathing test for 15 minutes (MSVC). Following those tests, a five week program of ventilatory muscle endurance training was undertaken. During the training procedure, the subjects were instructed to ventilate at 85% of their MVV_{15} four times a day. When the training intensity was attained, the quantity was increased by 5% on the next day. Following the training program, the experimental group showed an increase in MVV_{15}, as well as their endurance breathing; however, there was no significant increase in their VO_2 max.

In summary, it would appear that ventilatory muscle training in normal subjects has the capacity to increase both the strength and the endurance of the respiratory musculature, but a concurrent increase in VO_2 max has not yet been demonstrated as a result of this type of training.

Ventilatory Muscle Training in COPD Patients

For the past two decades, exercise training has been used in the rehabilitation of some COPD patients. This training was similar to that used in cardiac rehabilitation. Some of the results have been encouraging, particularly, the demonstration of improved work capacity in many of the cases. However, with this type of training there has not been evidence of improved pulmonary functions associated with the increased work tolerance (Brown and Wasserman, 1981; Belman and Mittman, 1980; Hale et al., 1978).

In the last few years, a number of studies have been directed at training the ventilatory muscles of COPD patients. Gimenez et al. (1977) described the effects of "directed breathing" training in the rehabilitation of patients with chronic lung disease. Their directed breathing technique was designed to alter the normal respiratory cycle by encouraging patients to inhale slowly and deeply through the nasal passageways, while exhaling through pursed lips utilizing the abdominal musculature. It was their theory that directed breathing increased the ventilatory efficiency by reducing the ventilatory rate while the patient maintained a similar minute volume. This increases the amount of alveolar ventilation per minute. The initial stage of their training program attempted to develop this directed breathing as the patient's new ventilatory pattern. The patients were continually coached by physical therapists during this time. Once a satisfactory breathing pattern was established, the patients moved into the second phase of the program which consisted of directed breathing for 30 minutes each morning and 30 minutes each night. In addition to the breathing exercises, they were to use this directed breathing technique during a mild exercise program which consisted of two 15 minute walking periods a day. The investigators evaluated respiratory and hemodynamic parameters before, during, and after directed breathing. They found that during directed breathing, there was improvement in the oxygen saturation of arterial blood and in the P_aO_2 and P_aCO_2, as well as a rather remarkable reduction in the respiratory

rate. They concluded that the improvement in directed breathing resulted from the improved ventilatory efficiency which increased the alveolar ventilation at a given minute volume. Secondly, they believed that this breathing technique faciliated bronchial drainage, thereby reducing resistance to airflow. They further indicated that the directed breathing pattern with appropriate practice could become the patient's normal ventilatory pattern.

In 1981, Casciari et al. examined the effects of breathing retraining on exercise tolerance in patients with severe COPD. These investigators selected 22 subjects and instructed them to exercise on a treadmill three times a week for six weeks. Following the initial six week training period, 12 of those subjects served as a control and continued with the present treadmill exercise program. The other 10 continued with the treadmill program but began a breathing retraining program to coincide with their aerobic training. The breathing retraining program in which this group participated was similar to that of Gimenez where the patients were encouraged to breathe deeply and slowly, exhaling through pursed lips. This breathing retaining program lasted for three weeks. Following the first six weeks of the training, the results of routine pulmonary function tests and exercise tests were similar for both the control group and the group that participated in the rebreathing training. Following the final three weeks of the study, performance of exercise tests were significantly higher on those subjects that received the breathing retraining when compared with the control.

Furthermore, those subjects undergoing the breathing retraining also demonstrated a reduction in their respiratory rate and an increase in their tidal volume and an increase in their P_aO_2 during exercise. The authors suggested that the breathing retraining, in conjunction with aerobic training, increased the work performance of patients with COPD.

During a six month rehabilitation program, including breathing exercises or a combination of breathing exercises and bicycle training, Sergysels et al. (1979) examined 20 patients suffering from COPD. The breathing exercises were, again, variations of the pursed lip breathing in which the respiratory rate was reduced while the depth of ventilation was increased. Following the six month training program, the authors reported a significant difference in the total lung capacity, vital capacity, and P_aCO_2 for those subjects engaged solely in the breathing program. They reported similar changes in the group with the breathing training and bicycling program. In addition, they claimed that those subjects who were engaged in the bicycle training also demonstrated significant changes in their P_aO_2 and symptom limited VO_2 max. The authors felt that the results were attributed to improved respiratory muscle strength and improved alveolar ventilation. They further stated that exercise training increases the ability to sustain higher workloads in patients with COPD.

There are two studies reporting the effects of resisted breathing training on ventilation in patients with COPD. Andersen

et al. (1979) studied the effects of resistive breathing training on 10 patients with long-standing severe COPD. These investigators were interested in determining when the diaphragm muscle fatigued. If the diaphragm fails as a generator of negative pressure during inspiration, "It will be sucked into the thoracic cavity when the pleural pressure falls owing to the action of the other inspiratory muscles." An examiner can witness this by the inward displacement of the abdomen during inspiration. Diaphragmatic fatigue was also evaluated through EMG signals. In this experiment, the subjects ventilated through a face mask which had inspiratory tubes of varying diameters. Initially, the inspiration was unloaded, but every two minutes a resisted load was added by using an inspiratory valve with a smaller diameter. The diameter was decreased every two minutes until evidence of diaphragm fatigue through paradoxical movements occurred. Once evidence of diaphragmatic fatigue was present, this load was established as the training load. These subjects were instructed to breathe into this device three times daily, at home, for as long as they could. At the end of the four week period, the subjects were re-evaluated. All patients improved by demonstrating higher resistance tolerance without signs of diaphragmatic fatigue following this training program. In addition, patients made subjective claims that this training helped them with their daily living and that they were able to be more active without shortness of breath.

In 1982, Sonne and Davis evaluated six patients with chronic obstructive pulmonary disease. These patients underwent a six week outpatient program using an inspiratory resistive device. This device had adjustable orifices which increased the inspiratory load of the patient. Pre- and post-tests of exercise performance were measured using the bicycle ergometer. Following the six week training program, the six patients demonstrated a 15% increase in their VO_2 max, a 37% increase in their maximum work rate, and a 17% increase in their minute volume during exercise. Furthermore, the patients were able to demonstrate a 56% increase in their respiratory muscle endurance. The authors reported that all the patients claimed increased ability to perform activities of daily living.

In 1979, Peress et al. conducted a pilot study on a group of adult subjects with obstructed lung disease. All had severe airway obstruction. Respiratory muscle endurance was assessed by determining the maximum sustained ventilatory capacity (MSVC) of these subjects over a 15 minute period under conditions of normocapnea. The subjects underwent a training program five days a week for one month. Training consisted of 25 minutes per day of maximal normocapnic hyperventilation to the point of exhaustion. At the termination of the training program, pulmonary function tests and blood gas analyses were performed. Their subjects demonstrated substantial improvement in MSVC and MVV_{15}. However, no other changes were noted in pulmonary functions or blood gases.

In 1980, Belman and Mittman examined the effects of a six week ventilatory training program on maximal sustained ventilatory capacity and exercise tolerance in patients with obstructive lung disease. They used a method similar to that of Peress et al. (1979). All patients were to perform two 15-minute periods of sustained maximum ventilation under conditions of normocapnea. Following the training period, the patients showed no significant changes in lung volume. There was an increase in the MSVC and in the oxygen consumption and heart rate recorded during the MSVC. Maximum exercise ventilation measured on a progressive ergometer test also increased for both leg and upper extremity exercises. Endurance time at a constant submaximal workload increased as did the maximum distance covered in a 12-minute walk. From these results, the authors concluded that ventilatory muscle training in patients with COPD improved ventilatory muscle performance and led to improved exercise tolerance.

In 1981, Pardy et al. examined an inexpensive home ventilatory training program utilizing 12 patients with moderate to severe air flow limitations. Again, the training of the inspiratory muscles was of an endurance type in which a state of normocapnea was maintained. Following the training program, all of the subjects showed increases in the maximum sustained ventilatory capacity. Seven of the 12 subjects increased their submaximal exercise performance and 6 of the subjects did not demonstrate improvement. Retrospectively, the investigators discovered that six of the seven patients showing improvement in submaximal exercise demonstrated signs of inspiratory

muscle fatigue through electromyographic changes on their pre-training submaximal exercise tests. Of the five patients who showed no improvement in exercise tolerance following the training, none demonstrated signs of inspiratory muscle fatigue on their submaximal exercise test prior to training. From this, the authors concluded that endurance training of inspiratory muscles of COPD patients is associated with improved exercise tolerance only in those individuals demonstrating inspiratory muscle fatigue during exercise.

Belman and Kendregan (1982) examined the effects of general physical training on ventilatory muscle endurance on patients with COPD. They selected 15 symptomatic COPD patients and had 7 of the patients train their lower extremities for 6 weeks, and 8 patients train their upper extremities. Before and after training, exercise tolerance, pulmonary function, and maximal sustained ventilatory capacities were evaluated. Following the training period, the subjects showed improvement in their ability to perform work; however, there was no significant change in spirometric values or in their MSVC. As a result, the authors concluded that non-ventilatory exercises do not improve ventilatory muscle performance.

CHAPTER III

METHODS

Purpose

The purpose of this study was to determine if specific pulmonary function and physical work capacity measures associated with COPD could be altered through the use of ventilatory, aerobic, or a combination of ventilatory and aerobic training.

Test Selection

The test items selected in this study fell into two general categories. The first were pulmonary function evaluations, and the second, a test of physical work capacity. The following pulmonary function tests were selected: forced vital capacity (FVC), and forced expiratory volume 1 second (FEV_1). The ratio of FEV_1 divided by FVC was reported as the forced expiratory volume for 1 second percent ($FEV_1\%$). The final test was maximum voluntary ventilation for 10 seconds (MVV_{10}).

The FVC was measured using a standard Warren-Collins Survey Spirometer, Model #06034. The subject assumed a sitting position, and a nose clip was applied. He/she was instructed to take a maximum inspiration and then told to exhale as much air as possible. The subject was then informed that the time was not a critical factor in performing this maneuver, but that we were interested in the maximum amount of air he/she could expire in one breath. After

the subject received the instructions and practiced the test procedure, the maneuver was performed. The subject made three attempts and the best maximal volume was recorded.

The FEV_1 was measured with the same Collins Spirometer. The subject was again placed in a sitting position and a nose clip was applied. This time he/she was instructed to perform a maximum inhalation and then instructed to forcefully exhale as much air as possible. The emphasis in this procedure was to exhale a maximum amount of air in as short a time as possible. The subject was able to practice this procedure, and once he/she felt comfortable with it, the subject was given three attempts. Again, the highest score was recorded as data for this study.

The observed $FEV_1\%$ was calculated by dividing the FEV_1 by the FVC (FEV_1/FVC) and was expressed as a percent. This value was then compared with the predicted value for normal subjects the same age, height, and sex. The formula for determining the predicted values for FVC and FEV_1 for normal subjects came from the Hewlett Packard Company's Pulmonary Functions/Vital Capacity Manual, HP/67/69, User's Library Program based on the work by Morris et al. (1971). The predicted $FEV_1\%$ was calculated in the same manner as the observed $FEV_1\%$.

The observed $FEV_{1\%}$ was then divided by the predicted $FEV_1\%$. This value is known as the $FEV_1\%\%$. Individuals whose $FEV_1\%\%$ was calculated at less than 1.0 were selected for subjects in this study.

Furthermore, all subjects selected for this study were ranked according to their FEV_1%% scores and were assigned to specific test groups, minimizing the differences between groups based on this test (Table 1).

TABLE 1. Rank Order of Subjects Based on Forced Expiratory Volume for One Second Percent/Percent and Group Assigments

Rank Order	%%	GP	Rank Order	%%	GP
1	99.4	AV	20	92.0	A
2	99.4	A	21	91.5	V
3	98.6	V	22	91.5	AV
4	97.7	V	23	91.5	C
5	97.7	AV	24	91.4	AV
6	97.6	A	25	91.0	A
7	97.5	C	26	90.7	V
8	97.3	A	27	90.0	C
9	96.7	V	28	90.6	V
10	96.7	AV	29	89.6	AV
11	96.4	C	30	89.5	A
12	96.2	AV	31	86.9	C
13	96.2	A	32	84.7	A
14	96.1	V	33	84.6	V
15	95.6	C	34	83.2	AV
16	94.7	V	35	83.0	AV
17	94.3	AV	36	74.1	A
18	94.0	A	37	73.9	V
19	93.4	C			

The subjects in the control group completed only the pre- and post-training tests.

The MVV_{10} was measured by collecting the subject's expired gas in a meterological balloon, and then passing it through a

Parkinson/Cowan CD 4 Dry Gas Meter. The subject was placed in a sitting position, and a nose clip was applied. The subject was instructed to breathe as fast as he/she could for a 10 second period. The expired gas passed through a Collins valve (Triple J High Velocity Valve #21015) and was collected in the meterological balloon. The subject had the opportunity to practice this maneuver. When he/she felt comfortable with this procedure and after an adequate rest period, the subject then performed the MVV_{10} procedure.

Relative humidity, barometric pressure, and temperature were recorded during all pulmonary functions tests. All values were standardized and recorded as volumes BTPS.

$$\text{Volume BTPS} = \text{Volume ATPS} \frac{273+37}{273+t} \frac{P_B - P_{H_2O}}{P - 47}$$

(Fox and Mathews, 1981)

The standard Balke Treadmill Test was used to evaluate physical work capacity (Table 2). This treadmill test consisted of walking at a constant speed of three miles per hour. The subject started at a 0% grade, and every 2 minutes the grade was increased by 2.5%. The treadmill in this study was an Avionic's Model E-100. The 3 mile per hour speed was calibrated to be 38 belt revolutions per minute. To insure that the subjects always walked at a constant velocity, the belt revolutions for a 15 second period were counted every time the treadmill grade was changed. The belt revolutions were counted, rather than relying on the treadmill's speed control for producing a constant velocity.

TABLE 2. Balke Treadmill Test

Phase	%	Grade	Minutes	HR
I	0 (standing)		0-1	--
II a	0 (walking)		1-2	--
b	0		2-3	--
c	0		3-4	--
III a	2.5		4-5	--
b	2.5		5-6	--
IV a	5		6-7	--
b	5		7-8	--
V a	7.5		8-9	--
b	7.5		9-10	--
VI a	10		10-11	--
b	10		11-12	--
VII a	12.5		12-13	--
b	12.5		13-14	--
VIII a	15		14-15	--
b	15		15-16	--
IX a	17.5		16-17	--
b	17.5		17-18	--
X a	20		18-19	--
b	20		19-20	--

During the Balke test, the subject wore a nose clip and continually ventilated through the Collins Triple J Valve. The subject was monitored on an Avionics electrocardiogram, Model 2900B with a 10 second heart rate strip taken at 45 to 55 seconds of each minute. Once a pulse rate of 150 was obtained, the subject continued to walk at that same work load for an additional 30 seconds and expired gas was collected in the meterological balloon. Then the volume of expired gas was measured with the Parkinson/Cowan Dry Gas Meter and reported as expired ventilatory volume for 30 seconds (VE_{30}).

The time of the test and the actual workload was recorded so that this ventilation measure could be repeated at the same time and at the same workload during the post-training test regardless of the subject's heart rate response.

Subjects

Initially the goal was to find 40 volunteer subjects to participate in this training program. There were three limitations established for the participants. First, their age range was to be between 20 and 45 years. Secondly, they were to have no known medical problems for which they were currently being treated. Finally, their FEV_1% had to be below the predicted value for normal subjects of similar age, height, and sex but they were not to demonstrate any clinical symptoms of COPD. In excess of 250 individuals were evaluated with pulmonary function tests before 40 subjects could be identified. These 40 volunteer subjects were informed about the type of training program that they would be engaged in and the commitment in time that participation in this study would entail. Informed consent was obtained, and the subjects were scheduled for their pre-training tests. Of the 40 subjects, only 37 completed the pre-training tests.

Test Groups

There were four groups in this study. A control group (C) and three training groups. The first training group participated in aerobic training (A), the second in ventilatory exercise (V),

and the third in a combination of aerobic training and ventilatory exercise (AV). This was a six week training study in which all participants were evaluated in pre- and post-training tests. Following the pre-training tests, the subjects were all ranked based on the scores of their $FEV_1\%\%$ which is the observed $FEV_1\%$ divided by the predicted $FEV_1\%$; they were then specifically assigned to test groups to minimize the differences between groups. The control group consisted of 7 participants and the other training groups consisted of 10 participants each (Table 1).

Training Apparatus and Procedures for Aerobic Training

Two bicycle ergometers were used in this study. One was a Monarch Model #7101 and the other was a Tenturi Model 65140. During the training period, the subject's pulse was continually monitored by a Sears Pulse Meter (Model #564-2951) that was attached to the subject's ear and gave a continual display of the exercising subject's pulse rate. The subjects were instructed to exercise for their assigned period of time within two beats (plus/minus) of their target heart rate. The subjects who participated in aerobic training followed the procedure as outlined in Table 3.

Training Apparatus and Procedure Used for Ventilatory Training

Ventilatory training was accomplished through the use of a rebreathing tube that was developed by this investigator for this study. The rebreathing tube was designed to permit the subjects

TABLE 3. Aerobic Training with Bicycle Ergometer Three Times Per Week

Week #1	Exercise Heart Rate (HR) - 100 beats/min. Time: 10 minutes x 2
Week #2	Exercise (HR) - 70% age adjusted maximum heart rate (AAMax)* Time: 15 minutes x 2
Week #3	Exercise HR - 70% AAMax Time: 25 minutes
Week #4	Exercise HR - 70% AAMax Time: 30 minutes
Week #5	Exercise HR - 75% AAMax Time: 30 minutes
Week #6	Exercise HR - 75% AAMax Time: 30 minutes

*AAMax = 220 beats/min.-subject's age.

to breathe at a rate and depth that exceeded their metabolic demands. The theory was that if an elevated level of CO_2 could be retained in the rebreathing tube, then the excessive respiratory rate would not cause substantial lowering of the $PaCO_2$, and therefore, the subject would not suffer from the common hyperventilation syndrome which occurs when a person is in a state of hypocapnia. Various sizes and shapes of rebreathing tubes were tested and evaluated. The design that appeared to be adequate for this study consisted of a rigid tube, 10 cm in diameter, with a variable length that would allow the tube's volume to equal the subject's vital capacity.

A diaphragm-type apparatus was placed on both ends of the tube. Small pieces of Collins tubing, 3 cm in diameter, were attached to both ends. On one end, a mouthpiece was inserted so that the subject could breathe through it (Figure 1). The subject would place the mouthpiece in his/her mouth and breathe quietly for approximately 15 seconds. That 15 second period was necessary to allow the concentration of carbon dioxide in the tube to substantially increase above the concentration of CO_2 in the atmosphere. Following that 15 second period, the subjects could then breathe rapidly and deeply for a prolonged period of time without demonstrating signs of the hyperventilation syndrome. Once the subject became skilled with this rebreathing tube, he/she followed the training schedule which is listed below (Table 4).

The test group which participated in the combination of aerobic training and ventilatory exercise followed the aerobic training program and ventilatory exercises of the preceeding two groups.

Statistics

A one-way analysis of variance (ANOVA) procedure was used on the pre-training scores for $FEV_1\%/\%$, $FEV_1\%$, MVV_{10}, HR and VE_{30} tests to determine whether there were any significant differences between the mean scores of the different groups or any of the tests prior to the training program (Gay, 1976). No significant differences appeared (P values ranged from .961 to .719); as a result, the same ANOVA procedure was applied to the post-training scores of all

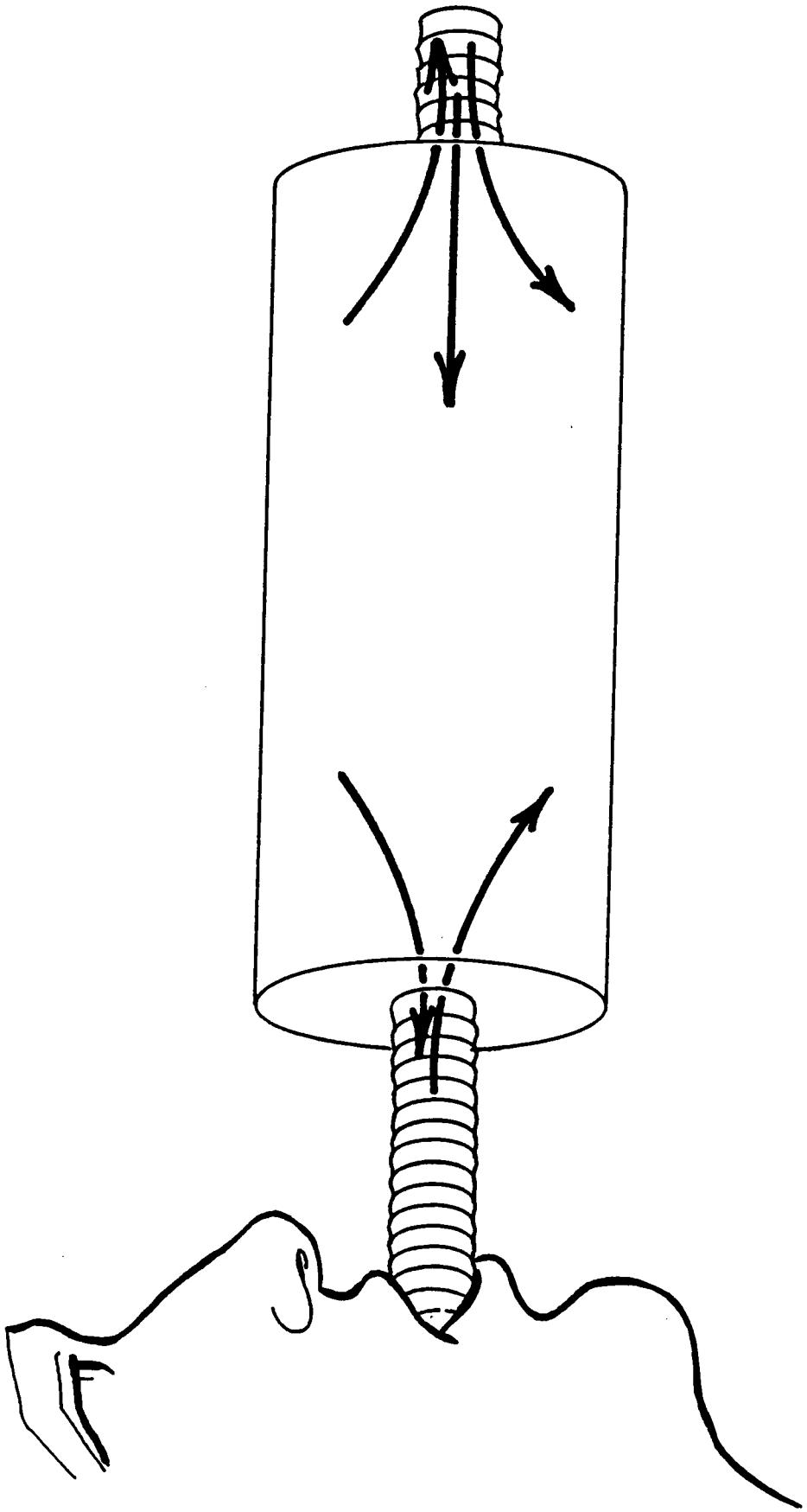


FIGURE 1. Re-breathing tube developed for ventilatory muscle training.

TABLE 4. Ventilation Training with Re-breathing Tube Five Times Per Week

Week #1	Day 1 Time: 5-minute set x 2 Ventilate: 15 seconds; Rest: 15 seconds Rest 1 minute between sets
	Day 2 Time: 5-minute set x 2 Ventilate: 45 seconds; Rest: 15 seconds Rest 1 minute between sets
	Days 3-5 Time: 5-minute set x 2 Ventilate: 50 seconds; Rest 10 seconds Rest 1 minute between sets
Week #2	Time: 5-minute set x 3 Ventilate: 55 seconds; Rest: 5 seconds Rest 1 minute between sets
Week #3	Time: 5-minute set x 3 Ventilate: 5 minutes Rest 1 minute between sets
Week #4	Time: 7-1/2-minute set x 2 Ventilate: 7-1/2 minutes Rest 1 minute between sets
Week #5	Time: 15 minutes Ventilate: 15 minutes
Week #6	Time: 15 minutes Ventilate: 15 minutes

groups to allow a comparison of the control group with the test group (Gay, 1976; H. H. Meyers, personal interview, January 7, 1984).

A Duncan Multiple Range Post Hoc Analysis was applied to any of the post-training tests where a significant difference appeared (Gay, 1976). A probability value of .05 or less was used to make generalizations.

CHAPTER IV

RESULTS

On the basis of the pre-training scores of forced expiratory volume percent/percent ($FEV_1\%/\%$), 37 subjects were assigned to one of four test groups: a control (C), aerobic training (A), ventilatory training (V), and a combination of aerobic and ventilatory training (AV), in a manner which minimized between group differences (see Table 1, page 40). An analysis of variance (ANOVA) was performed on the pre-training scores for $FEV_1\%/\%$, and no significant differences were found between the groups (Table 5).

TABLE 5. Forced Expiratory Volume for 1 Second Percent/Percent: One Way ANOVA Between Groups Based on Pre-Training Results

Source	D.F.	Sum of Squares	Mean Squares	F	Sig.
Between Groups	3	12.0993	4.0332	0.097	0.9611
Within Groups	33	1370.0712	41.5.73		
Total	36	1382.1708			

Four additional tests were performed on all subjects in the pre-training phase of the study. The first test measured forced expiratory volume for 1 second percent ($FEV_1\%$), which was determined by dividing the subjects' forced expiratory volume for 1 second

(FEV_1) by their forced vital capacity (FVC). The second test was a 10 second maximum voluntary ventilation (MVV_{10}), the third was the heart rate (HR) response at a fixed submaximal work load, and the fourth was the pulmonary ventilation measured in the last 30 seconds (VE_{30}) of the submaximal work test at a heart rate of 150. A one way analysis of variance was performed on each of these test items prior to the training program and no significant difference existed among the groups in any of these test items (see Appendix, pages 82-83).

Following the training period, the same battery of tests was administered to all subjects. An ANOVA procedure was then applied to the post-training scores for all the tests. In those tests where significant differences were found, the Duncan Multiple Range Post Hoc Evaluation at the .05 level was applied.

Table 6 shows the post-training means and standard deviations of the test groups in FEV_1%%. An ANOVA of the post-training scores on FEV_1%% was performed and no significant difference occurred between the groups (Table 7).

TABLE 6. Forced Expiratory Volume for 1 Second Percent/Percent: Pre- and Post-Training Scores

	Control	Aerobic	Ventilatory	Aerobic/Ventilatory
Pre	.931 ± .037	.918 ± .073	.915 ± .075	.923 ± .057
Post	.948 ± .077	.911 ± .085	.923 ± .087	.921 ± .101

Values are $\bar{X}$ and SD.

TABLE 7. Forced Expiratory Volume for 1 Second Percent/Percent: One Way ANOVA Between Groups Based on Post-Training Scores

Source	Sum of Square	D.F.	Mean Square	F	Sig
Between Groups	.0061	3	.0020	.258	.8553
Within Groups	.2619	33	.0079		
Total	.2680	36			

Table 8 shows the post-training means and standard deviations for the test groups in $FEV_1\%$. An ANOVA was performed on the post-training scores of $FEV_1\%$ and no significant differences occurred between the groups (Table 9).

TABLE 8. Forced Expiratory Volume for 1 Second: Pre- and Post-Training Scores

	Control	Aerobic	Ventilatory	Aerobic/Ventilatory
Pre	.73 $\pm$ .07	.71 $\pm$ .11	.72 $\pm$ .05	.72 $\pm$ .05
Post	.76 $\pm$ .07	.78 $\pm$ .07	.77 $\pm$ .06	.74 $\pm$ .08

Values are $\bar{X}$ and SD and are expressed in liters.

TABLE 9. Forced Expiratory Volume for 1 Second: One Way ANOVA Between Groups on Post-Training Scores

Source	D.F.	Sum of Squares	Mean Squares	F	Sig.
Between Groups	3	.0092	.0031	.559	.6204
Within Groups	33	.1690	.0051		
Total	36	.1782			

Table 10 shows the post-training means and standard deviations for MVV_{10} of the four groups. An ANOVA was performed on the post-training scores for MVV_{10} and a significant difference was found between the groups (Table 11). In MVV_{10} the post-training scores of the ventilatory and aerobic/ventilatory groups were different from the aerobic group (Table 12). Figure 2 provides a comparison of the pre- and post-training mean scores in MVV_{10} for all the groups.

TABLE 10. Maximum Voluntary Ventilation in Liters for 10 Seconds: Pre- and Post-Training Scores

	Control	Aerobic	Ventilatory	Aerobic/Ventilatory
Pre	21.75 $\pm$ 4.22	19.96 $\pm$ 6.23	22.39 $\pm$ 7.60	22.78 $\pm$ 4.31
Post	21.43 $\pm$ 4.69	20.24 $\pm$ 6.44	26.60 $\pm$ 7.36	25.89 $\pm$ 3.54

Values are $\bar{X}$ and SD.

TABLE 11. Maximum Voluntary Ventilation for 10 Seconds: One Way ANOVA Between Groups Based on Post-Training Scores

Source	Sum of Square	D.F.	Mean Square	F	Sig.
Between Groups	287.7039	3	95.9013	2.862	.0516
Within Groups	1105.7782	33	33.5084		
Total	1393.4821	36			

TABLE 12. Maximum Voluntary Ventilation for 10 Seconds: Duncan Multiple Range at .05 Level Reported in Similar Subsets

Subset 1	A(20.24)	C(21.43)	
Subset 2	C(21.43)	A/V(25.89)	V(26.60)

.05 level

	C	A	V	A/V
C	x	x	x	x
A	x	x		
V	x		x	x
A/V	x		x	x

(x indicating similarity).

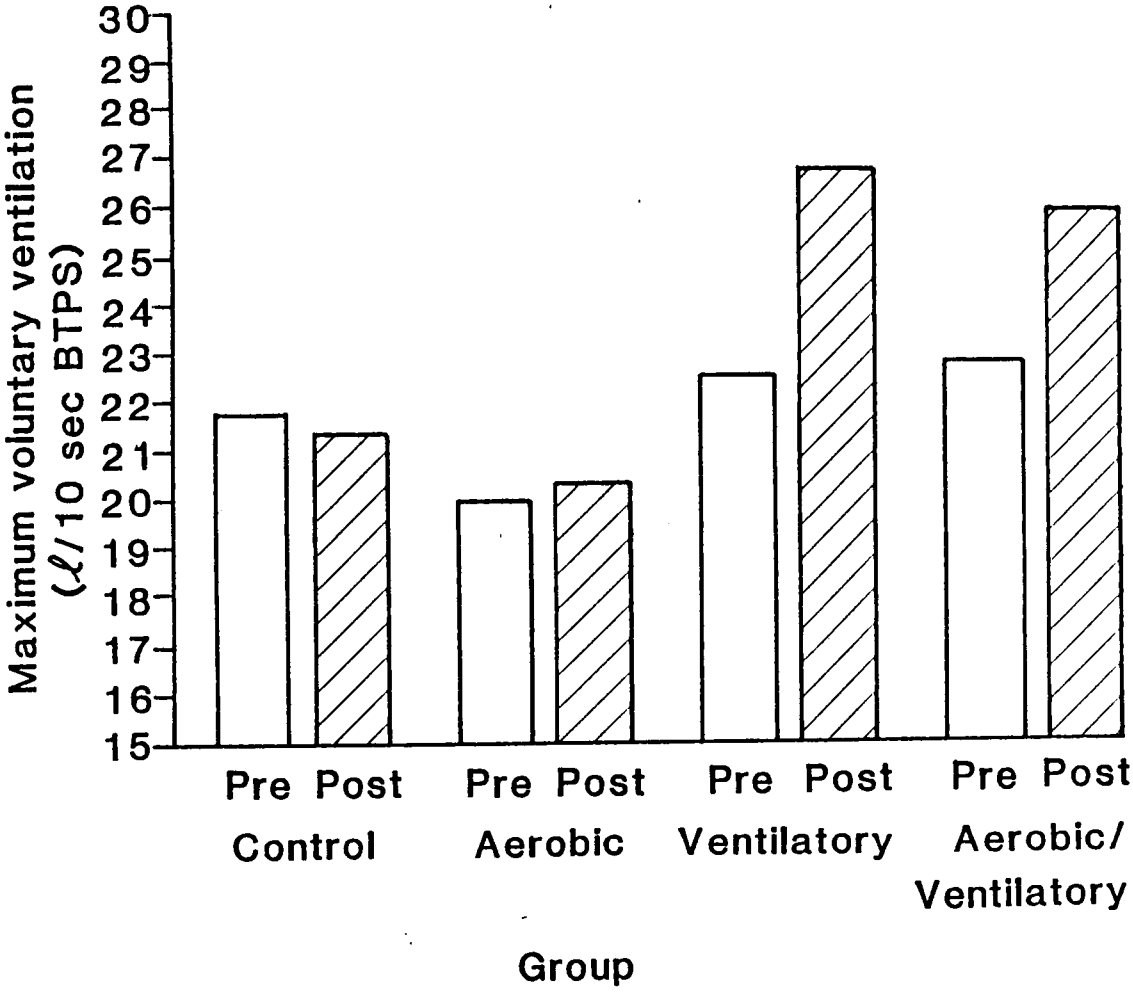


FIGURE 2. Pre- and post-training mean scores for maximum voluntary ventilation in liters for 10 seconds.

Table 13 shows the post-training means and standard deviations for HR of the four groups. An ANOVA was performed on the post-training HR scores and a significant difference existed between the groups (Table 14). In HR the aerobic group was significantly different from the control and ventilatory groups, and the aerobic/ventilatory group was significantly different from the ventilatory group (Table 15). Figure 3 provides a comparison of the pre- and post-training mean scores in HR for all groups.

TABLE 13. Heart Rate in Beats Per Minute: Pre- and Post-Training Scores

	Control	Aerobic	Ventilatory	Aerobic/Ventilatory
Pre	154.4 ± 3.7	155.5 ± 5.1	155.0 ± 8.7	153.7 ± 5.9
Post	155.4 ± 13.1	142.2 ± 5.7	158.7 ± 17.8	145.5 ± 11.5

Values are $\bar{X}$ and SD.

TABLE 14. Heart Rate: One Way ANOVA Between Groups Based on Post-Training Scores

Source	D.F.	Sum of Squares	Mean Squares	F	Sig.
Between Groups	3	1809.6620	603.2207	3.956	.0169
Within Groups	31	4727.3143	150.4940		
Total	34	6536.9763			

TABLE 15. Heart Rate: Duncan Multiple Range at .05 Level Reported as Similar Subsets

Subset 1	A(142.1)	A/V(145.5)
Subset 2	A/V(145.5)	C(157.4)
Subset 3	C(157.4)	V(158.7)

.05 level

	C	A	V	A/V
C	x		x	x
A		x		x
V	x		x	
A/V	x	x		x

(x indicating similarity.)

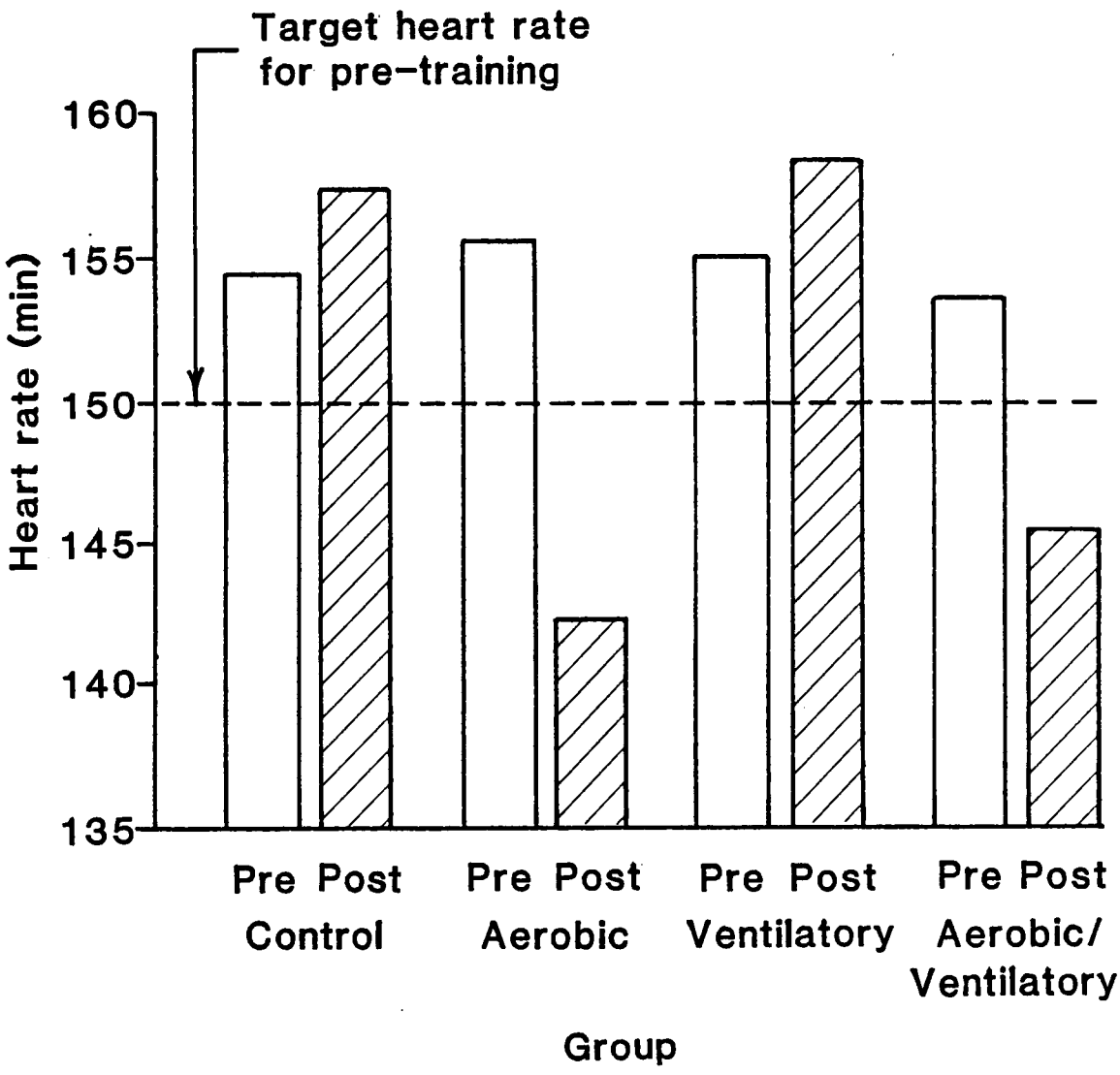


FIGURE 3. Pre- and post-training mean scores of heart rate response to submaximal exercise.

Table 16 shows the post-training means and standard deviations for VE_{30} of all the groups. An ANOVA was performed and no significant difference appeared between groups (Table 17).

TABLE 16. Pulmonary Ventilation for 30 Seconds in Liters: Pre- and Post-Training Scores

	Control	Aerobic	Ventilatory	Aerobic/Ventilatory
Pre	54.71 ± 12.24	53.27 ± 11.94	57.93 ± 16.26	53.19 ± 15.95
Post	61.14 ± 18.67	46.53 ± 10.99	60.47 ± 19.40	52.78 ± 11.68

Values are $\bar{X}$ and SD.

TABLE 17. Pulmonary Ventilation for 30 Seconds in Liters: One Way ANOVA Between Groups Based on Post-Training Scores

Source	D.F.	Sum of Squares	Mean Squares	F	Sig.
Between Groups	3	1258.1597	419.3866	1.846	.1594
Within Groups	31	7043.3204	227.2039		
Total	34	8301.4801			

CHAPTER V

DISCUSSION

Early intervention may offer the most optimistic results in altering or slowing the process of COPD according to Hale et al. (1978) and Ryan (1981). The purpose of this study was to determine if specific pulmonary function and physical work capacity measures associated with COPD could be altered through ventilatory exercises, aerobic training, or a combination of these programs in subjects with preclinical COPD. The subjects were divided into four groups: a control, aerobic training, ventilatory training, and a combination of aerobic/ventilatory training. Pre- and post-training measures of $FEV_1\%/\%$, $FEV_1\%$, MVV_{10}, submaximal HR and VE_{30} at a submaximal heart rate were evaluated. The post-training results revealed significant differences in the post-training scores in only MVV_{10} and HR.

Maximum Voluntary Ventilation

In MVV_{10}, the aerobic group had the lowest pre-training score (20.24ℓ) and was significantly different from the post-training scores of the ventilatory group (26.60ℓ) and aerobic/ventilatory group (25.89ℓ) at the .05 level. The control group had a slightly higher post-training score (21.43ℓ) than the aerobic group and was not significantly different from the ventilatory or aerobic/ventilatory groups. Modest gains were present between the pre- and

post-training scores in MVV_{10} in the ventilatory group (18.8%) and in the aerobic/ventilatory group (13.6%) as opposed to a minimal increase in the aerobic group (3.2%) and a decline in the control group (1.4%).

Leith and Bradley (1976) demonstrated an increase in MVV_{15} through exhaustive endurance training of the ventilatory muscles of normal subjects. Peress et al. (1979), Pardy et al. (1981), and Belman and Mittman (1980) evaluated the effects of exhaustive ventilatory endurance training in subjects with COPD and were also able to demonstrate an increase in MVV_{15}. In this study, however, the ventilatory training was not designed to be exhaustive in nature; furthermore, it required no sophisticated equipment. On the contrary, the subject was instructed to breathe at a comfortable rate for 15 minutes. It was felt that this type of program would be one in which a true COPD patient could participate for prolonged periods of time and would not require the motivation demanded in more exhausting training programs. In many cases, the subjects who used our respiratory device indicated that they could practice while they were watching the news or while they were reading. They indicated that it was not a particularly unpleasant experience, and that they would be willing to participate in such a program for prolonged periods of time if it would benefit them. The fact that the subjects did not consider this a vigorous program is advantageous, for more severely impaired COPD patients could participate in such a program without undue duress. Even though this ventilatory program

was not exhausting, the subjects did show a mean increase in their MVV_{10}. This may be surprising to some readers because MVV_{10} is an intense activity. Improvement in MVV_{10} would be expected as a result of strength training rather than the non-exhaustive endurance training employed.

Heart Rate Response

The aerobic training in this study consisted of peddling a bicycle ergometer for an assigned period of time at a target heart rate. The pre- and post-training submaximal exercise tests in this study were performed on the treadmill. This method was used to avert the speculation that a decrease in heart rate, following the training program, could be the result of increased efficiency on the training equipment rather than as a result of improved efficiency of the cardiorespiratory system. The post-training mean score for heart rate of the ventilatory and control groups were 158.7 and 157.4, respectively. The mean post-training HR score for the ventilatory group was significantly different from that of the aerobic group (142.1) and the aerobic/ventilatory group (145.5). Statistically the control group was different from the aerobic group but not the aerobic/ventilatory group.

Pierce et al. (1964), Nicholas et al. (1970), and Mertens et al. (1978) demonstrated the effects of walking programs on sedentary subjects with COPD. All the investigators reported improved work capacity without associated improvements in pulmonary functions.

Alison et al. (1981) evaluated the effects of a 12 week progressive bicycle program on patients with COPD and found that there were significant increases in the 12 minute walk scores in their subjects following the training.

That there was not a significant difference in the post-training heart rate scores between the aerobic/ventilatory group and the control group might be due to the fact that the exercise period may not have been long or intense enough, or more likely be a function of the relatively small numbers of subjects.

Pulmonary Function Measures

Improved efficiency at a given submaximal workload is demonstrated by a reduction in HR and pulmonary ventilation (VE_{30}). Although no significant differences occurred in the post-training score for VE_{30} between groups, it is interesting to note that the control group increased their VE_{30} by 11.7% and the ventilatory group increased their VE_{30} by 4.4%. Both of these groups demonstrated a mean increase in their post-training HR scores. The aerobic group had a decrease in their post-training HR scores and demonstrated a 14.5% decrease in their post-training VE_{30}. The aerobic/ventilatory group also had a decrease in their post-training HR but showed only a 0.7% decrease in VE_{30}. A possible explanation for the minimal decrease in VE_{30} for the aerobic/ventilatory group became apparent after an interview with some of these subjects. It was discovered that they thought an increase in VE_{30} was expected as a result of

their ventilatory training. Therefore, the possibility exists that a few of these subjects may have attempted to improve our results.

Pulmonary function measures often used in the diagnosis of COPD are FVC, FEV_1, and FEV_1% (Black, 1982; Ryan, 1981; Epler et al., 1980). There was no evidence to suggest that beneficial changes occurred in these measures as a result of participation in the ventilatory training used in this study. These results are similar to those of Peress et al. (1979) and Belman and Mittman (1980) who used different forms of ventilatory muscle training but failed to show improvement in these pulmonary function measures.

Conclusions

Within the limitations of this study the following conclusions are made.

1. The aerobic training used in this study resulted in a reduction in HR while performing a submaximal workload but this training did not result in a change in pulmonary function.
2. The ventilatory muscle training used in this study demonstrated an increase in MVV_{10} independent of any HR response to submaximal exercise.
3. Combining the aerobic bicycle training and the ventilatory muscle training did not result in a more significant increase in MVV_{10} than did the ventilatory muscle training alone or a greater decrease in the HR during submaximal exercise than did the aerobic bicycle training alone.

Recommendations

The ventilatory device developed for the ventilatory muscle training in this study did produce an increase in MVV_{10} in subjects with mild, pre-clinical COPD. The same type of training program should be attempted on clinical patients to determine if the same type of results are possible.

Pulmonary ventilation is thought to play a role in limiting maximal aerobic performance in COPD patients. It would be beneficial to design a training study to determine if a COPD subject's maximum aerobic performance could be increased through the ventilatory muscle training program used in this study, alone or in combination with some form of aerobic training.

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APPENDIX

INFORMED CONSENT

DESCRIPTION OF PROJECT: The purpose of this program is to determine whether or not the pulmonary functions of individuals demonstrating mild airway obstruction can be improved following specific training programs. All participants will be required to take pre- and post-training tests of pulmonary function and physical work capacity (involving treadmill walking at moderate rates of speed). Furthermore, participants will be divided into specific training groups and may be involved in ventilatory exercises, aerobic training, or a combination of both. The duration of this study is approximately six (6) weeks.

All participants will be given ample time to become acquainted with the testing and training equipment prior to the beginning of the study. Participants have been selected for this experimental program based on a pulmonary functions test, FEV_1 (forced expiratory volume one second) that was less than the predicted value for normal individuals of similar age, height, and sex.

POTENTIAL RISKS AND DISCOMFORTS: The rate and intensity of physical work required in both the test and training phases of this study should be considered moderate and of minimal risk. There are, however, risks associated with any type of physical exercise or training. These risks include muscle soreness, joint pain, headache, shortness of breath, and heart attack. The possibility of any of these complications in this study are considered minimal.

PARTICIPANTS' RIGHTS: As a participant in this study, you should be aware of the following:

1. You are entitled to ask any questions involving the testing and training procedures to be used in this program, and to ask any questions about possible risks involved in participating in this activity, now or in the future.

2. In the event of presentation or publication of some or all of this data, you are assured anonymity.

3. You are volunteering to participate in this study. However, YOU MAY WITHDRAW YOUR CONSENT AND DISCONTINUE PARTICIPATION AT ANY TIME without explanation or fear of penalty or prejudice towards you.

I HAVE READ THE FOLLOWING STATEMENT AND I UNDERSTAND THE NATURE OF THE PROJECT AND MY ROLE AS A PARTICIPANT.

_____ signature _____ date

INVESTIGATOR:

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TABLE 18. Forced Expiratory Volume for 1 Second, Percent: One Way ANOVA for Differences Between Groups Based on Pre-Training Results

Source	D.F.	Sum of Squares	Mean Squares	F	Sig.
Between Groups	3	.0023	.0008	.143	.9334
Within Groups	33	.1794	.0054		
Total	36	.1818			

TABLE 19. Maximum Voluntary Ventilation for 10 Seconds: One Way ANOVA for Differences Between Groups Based on Pre-Training Results

Source	D.F	Sum of Squares	Mean Squares	F	Sig.
Between Groups	3	46.8039	15.6013	0.450	0.7190
Within Groups	33	1144.0226	34.6674		
Total	36	1190.8265			

TABLE 20. Heart Rate: One Way ANOVA for Differences Between Groups Based on Pre-Training Results

Source	D.F.	Sum of Squares	Mean Squares	F	Sig.
Between Groups	3	17.5741	5.8580	0.156	0.9252
Within Groups	31	1166.3143	37.6230		
Total	34	1183.8884			

TABLE 21. Pulmonary Ventilation for 30 Seconds: One Way ANOVA for Differences Between Groups Based on Pre-Training Results

Source	D.F.	Sum of Squares	Mean Squares	F.	Sig.
Between Groups	3	126.2593	42.0864	0.206	0.8912
Within Groups	31	6320.9128	203.9004		
Total	34	6557.1721			

TABLE 22. Forced Expiratory Volume for 1 Second Percent/Percent: Raw Scores Pre- and Post-Training

Subjects	Control		Aerobic		Ventilatory		Aerobic/Ventilatory	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	.975	1.065	.994	.944	.986	.978	.994	.991
2	.964	.966	.976	.922	.977	.944	.977	.906
3	.956	.955	.973	1.012	.967	.970	.967	1.006
4	.934	1.020	.962	.967	.961	1.001	.962	1.118
5	.915	.863	.940	.934	.947	1.002	.943	.939
6	.907	.906	.920	.877	.915	.937	.915	.939
7	.809	.860	.910	.871	.907	.915	.914	.885
8	--	--	.895	1.013	.906	.936	.896	.856
9	--	--	.847	.836	.846	.795	.832	.818
10	--	--	.741	.733	.739	.743	.830	.791

TABLE 23. Forced Vital Capacity in Liters: Raw Scores Pre- and Post Training

Subjects	Control		Aerobic		Ventilatory		Aerobic/Ventilatory	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	3.79	3.76	2.70	2.75	4.59	4.63	4.01	4.11
2	4.23	4.02	4.43	4.63	4.86	5.08	5.07	5.26
3	6.02	5.99	4.93	4.73	5.39	5.43	4.72	4.77
4	4.29	4.45	4.03	4.16	3.80	3.60	4.67	4.69
5	5.83	6.01	4.84	5.15	4.66	4.79	3.94	3.91
6	5.19	5.08	3.92	3.95	4.12	4.57	5.14	5.33
7	4.09	4.20	4.10	4.17	3.12	3.41	4.73	4.92
8	--	--	4.08	4.65	3.38	3.46	5.86	5.70
9	--	--	2.75	2.91	3.39	3.39	4.15	4.17
10	--	--	6.48	6.14	2.83	2.99	4.65	4.92

TABLE 24. Forced Expiratory Volume for 1 Second in Liters: Raw Scores Pre- and Post Training

Subjects	Control		Aerobic		Ventilatory		Aerobic/Ventilatory	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	2.98	3.23	2.13	2.06	3.52	3.52	3.16	3.23
2	3.44	3.28	3.39	3.35	3.70	3.74	3.80	3.66
3	4.36	4.33	3.75	3.75	3.99	4.03	3.67	3.86
4	3.27	3.71	3.04	3.16	2.76	2.73	3.59	3.81
5	4.12	4.03	3.66	3.87	3.56	3.88	2.95	3.47
6	3.17	3.68	2.83	2.72	3.02	3.22	3.69	3.93
7	2.92	2.96	2.99	2.91	2.31	2.52	3.46	3.48
8	--	--	2.91	3.67	2.41	2.55	3.92	3.76
9	--	--	1.84	1.92	2.29	2.15	2.81	2.77
10	--	--	2.72	3.49	1.67	1.78	2.93	3.12

TABLE 25. Forced Expiratory Volume for 1 Second Percent: Raw Scores Pre- and Post-Training

Subjects	Control		Aerobic		Ventilatory		Aerobic/Ventilatory	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	.786	.859	.788	.749	.766	.760	.788	.786
2	.813	.816	.765	.724	.761	.736	.749	.696
3	.724	.723	.761	.792	.740	.742	.777	.809
4	.762	.833	.754	.759	.726	.758	.769	.812
5	.707	.670	.756	.751	.764	.810	.749	.887
6	.601	.724	.722	.689	.717	.725	.718	.737
7	.714	.705	.729	.698	.713	.739	.731	.707
8	--	--	.713	.789	.713	.737	.669	.660
9	--	--	.669	.660	.675	.634	.677	.664
10	--	--	.420	.568	.590	.595	.630	.634

TABLE 26. Maximum Voluntary Ventilation for 10 Seconds in Liters: Raw Scores Pre- and Post-Training

Subjects	Control		Aerobic		Ventilatory		Aerobic/Ventilatory	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	21.19	21.94	9.81	9.65	33.27	39.96	18.60	21.07
2	28.00	25.47	23.24	23.17	29.82	29.85	24.55	29.22
3	21.81	24.42	26.99	29.98	26.44	32.59	21.12	24.87
4	23.10	25.50	25.00	22.80	27.00	28.91	25.42	28.04
5	19.40	13.55	28.88	28.60	26.11	29.93	19.97	24.05
6	24.31	22.81	14.25	14.44	24.92	24.02	25.91	26.33
7	14.44	16.35	17.98	18.76	14.39	19.44	18.35	22.83
8	--	--	16.24	21.19	14.61	25.85	31.62	32.58
9	--	--	14.91	13.54	16.22	21.62	18.32	22.39
10	--	--	22.29	20.31	11.15	13.84	23.83	27.57

TABLE 27. Heart Rate In Beats Per Minute: Raw Scores Pre- and Post-Training

Subjects	Control		Aerobic		Ventilatory		Aerobic/Ventilatory	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	150	150	165	140	155	180	155	150
2	160	180	160	150	151	171	156	162
3	155	160	152	140	155	168	152	120
4	154	158	154	145	--	--	158	145
5	150	160	154	148	160	156	160	140
6	154	136	156	140	158	160	140	136
7	158	158	160	138	165	160	154	150
8	--	--	156	142	160	155	152	154
9	--	--	150	131	136	120	160	150
10	--	--	148	148	--	--	150	148

TABLE 28. Pulmonary Ventilation for 30 Seconds at a Submaximal Workload in Liters: Raw Scores Pre- and Post-Training

Subjects	Control		Aerobic		Ventilatory		Aerobic/Ventilatory	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	53.25	51.04	27.07	35.40	66.26	87.97	39.90	52.79
2	51.48	82.35	52.99	51.48	73.20	88.37	67.20	57.68
3	60.73160	71.57	70.77	66.26	73.68	62.89	45.60	40.90
4	44.37	42.90	52.11	37.80	73.42	--	52.07	51.49
5	79.32	86.39	59.73	52.40	41.07	43.72	66.32	56.40
6	49.66	41.38	67.63	62.10	73.68	62.89	80.15	74.62
7	44.15	52.44	53.19	42.35	51.13	50.35	45.92	47.09
8	--	--	49.57	42.59	52.83	52.26	57.59	50.36
9	--	--	51.30	37.02	31.57	35.19	23.44	32.55
10	--	--	48.34	37.89	42.89	--	53.70	63.91

VITA

Peter George Kramer was born in Coral Gables, Florida on February 15, 1945. He attended the public schools in New Brunswick, New Jersey and graduated from New Brunswick High School in June, 1963. The following September he entered Springfield College, Springfield, Massachusetts and in June, 1967 he received a Bachelor of Science degree in Physical Education. In January, 1969 he received a Master of Science Degree in Adapted Physical Education from Springfield College.

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