To the Graduate Council:

I am submitting herewith a thesis written by Don Whitford Morgan entitled "Effects of Respiratory Muscle Endurance Training on Maximal Oxygen Uptake in Untrained Men and Women." I have examined the final copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Physical Education.

Hugh G. Welch

Hugh G. Welch, Major Professor

We have read this thesis
and recommend its acceptance:

Edward T. Howley

B. D. Franks

Accepted for the Council:

L. Evans Tash

Vice Chancellor
Graduate Studies and Research

EFFECTS OF RESPIRATORY MUSCLE ENDURANCE TRAINING ON MAXIMAL OXYGEN UPTAKE IN UNTRAINED MEN AND WOMEN

A Thesis

Presented for the

Master of Science

Degree

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ABSTRACT

The purpose of this study was to determine the effects of respiratory muscle endurance training on maximal oxygen uptake in untrained men and women. Twelve volunteers (six male and six female) were placed into either an experimental or control group. Both groups completed a 15-second MVV test, an endurance breathing test, and a maximal oxygen uptake test. Following these tests, the experimental group underwent a strenuous five-week training program specifically designed to increase the ventilatory endurance of the breathing muscles. Every fifth day, measurements of 15-second MVV and endurance breathing time were taken. After the training period was completed, both groups repeated the three pre-test tasks. The experimental group showed significantly greater changes than the control group in 15-second MVV ($p<0.005$), endurance breathing time ($p<0.0005$), and peak ventilation attained during the maximal oxygen uptake test ($p<0.01$). There were no significantly different changes in maximal oxygen uptake ($.10>p>.05$); however, the experimental group showed a slight increase while the control group showed a decrease. These results indicate that ventilatory endurance is enhanced by a rigorous program of respiratory muscle endurance training. Investigation of the role that respiratory muscle training plays in maintaining acid-base balance during maximal aerobic work may prove helpful in understanding performance limitation in humans.

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CHAPTER I

INTRODUCTION

The ability to sustain great breathing efforts has not been thought to play a major role in limiting maximal aerobic performance in humans. Evidence shows that the peak ventilation during maximal exercise never exceeds the maximum ventilatory volume (Freedman, 1970; Shepherd, 1967; Tenney and Reese, 1968). Consequently, researchers have tended to dismiss the influence of ventilation as a limiting factor in exercise (Asmussen, 1965; Cotes, 1963; Demedts and Anthonisen, 1973; Holmgren and Linderholm, 1958; Stenberg, Ekblom, and Messin, 1966). In addition, the maintenance of normal PO_2 levels in arterial blood during heavy exercise further diminishes the importance of attaining maximal breathing efforts (Asmussen and Nielsen, 1958; Holmgren and Linderholm, 1958; Hughes et al., 1968; Taylor and Rowell, 1974).

However, the role of breathing as a limiting factor in exercise has not been totally discounted (Ouellet et al., 1969; Roussos and Macklem, 1977; Tenney and Reese, 1968). During intense, aerobic activity, non-respiratory work may be limited due to an increased oxygen cost of breathing in excess of the added oxygen intake achieved by maximal ventilatory efforts (Hughes et al., 1968; Margaria et al., 1960; Otis, 1954; Otis et al., 1950; Shepherd, 1966). Another consequence of the decreased availability of oxygen under these conditions

may be a predisposition of the respiratory muscles to fatigue (Levison and Cherniack, 1968; Macklem and Roussos, 1977; Roussos and Macklem, 1977). Martin and Stager (1981) suggest that in normal individuals, ventilatory muscle fatigue "would most likely occur when $\dot{V}_E$ is very high and thus most effective in maintaining PO_2 and pH homeostasis in arterial blood." If this is true, then increased ventilatory strength and endurance may enhance aerobic performance.

Recently, Bradley and Leith (1978) and Leith and Bradley (1976) showed that a program of respiratory muscle training increased both the strength and aerobic capacity of the breathing muscles. However, the relationship between respiratory muscle training and maximal aerobic performance has been examined in only one study, that published in abstract form (Haas and Haas, 1981); the authors found that respiratory muscle strength training did not significantly increase maximal oxygen uptake ($\dot{V}O_2$ max).

Endurance training of a particular intensity, duration, and frequency performed by untrained individuals can elevate $\dot{V}O_2$ max (Knehr et al., 1942; Robinson and Harmon, 1941; Saltin et al., 1968). No attempt has been undertaken to determine what effect respiratory muscle endurance training may have upon maximal oxygen uptake. It is known that $\dot{V}O_2$ max is an important factor in physical work capacity (Robinson et al., 1937; Saltin and Åstrand, 1967; Taylor et al., 1955). If ventilatory muscle endurance training increases this aerobic measure, then performance may be subsequently improved.

The purpose of this study, therefore, was to determine the effects of respiratory muscle endurance training on maximal oxygen uptake in untrained men and women.

CHAPTER II

REVIEW OF LITERATURE

Introduction

The pertinent review of the literature may be classified under six major categories. They are: 1) effects of ventilatory muscle training programs on normal individuals, 2) effects of ventilatory muscle training programs on diseased individuals, 3) effects of respiratory stress on animals, 4) effects of non-specific training programs on the ventilatory muscle responses in normal individuals, 5) respiratory muscle fatigue as a limiting factor in exercise, and 6) exercise ventilation.

Effects of Ventilatory Muscle Training Programs on Normal Individuals

In the first of two studies, Leith and Bradley (1976) measured respiratory muscle strength and endurance in normal individuals before and after a five-week training program. Four subjects, training for respiratory muscle strength, performed static maximal inspiratory and expiratory maneuvers against blocked airways. Four subjects, training for respiratory muscle endurance, executed a number of voluntary normocarbic hyperpnea runs to exhaustion, defined as "an inability to match a ventilatory target." Four subjects served as controls. As a result of the training program, the strength trainers increased inspiratory and expiratory pressure maximums, vital capacity, and total lung capacity. On the other hand, the endurance trainers increased

their maximal ventilatory volume (MVV) by 14% and were able to sustain 96% of their original MVV for a total of 15 minutes, as opposed to 81% prior to training.

In a subsequent experiment, Bradley and Leith (1978) examined the relationship between ventilatory muscle training and the oxygen cost of sustained hyperpnea. Following the protocol of their earlier study, they reported that the endurance trainers increased by 19% the level of hyperpnea that they could endure for 7 to 15 minutes. Also, the oxygen consumption of the respiratory muscles during hyperpnea rose by 67%, indicating a greater aerobic capacity. Fifteen weeks after the training period, ventilatory endurance was remeasured in three endurance trainers, two controls, and one strength trainer. The two controls and one strength trainer showed small changes in endurance capability. The three endurance trainers, however, lost 50% of their gains in ventilatory capacity and oxygen consumption.

Haas and Haas (1981) investigated the effects of inspiratory muscle resistive exercise training in nine healthy subjects. Each subject attempted to maintain 85% of the maximal inspiratory pressure at the mouth (Pi max) during two sets of three trials until fatigue occurred. The training regimen lasted 16 days. Results from the study indicated that strength training of the respiratory muscles increased the values of Pi max and MVV by 29% and 11%, respectively. In addition, the subjects were able to sustain Pi max for 20 breaths, as compared to only one or two breaths before training. A graded exercise treadmill test that measured "maximally tolerated limits" was also performed pre- and post-training; this was done to determine whether a

relationship existed between resistive training of the respiratory muscles and $\dot{V}O_2$ max. The authors found no post-training increase in $\dot{V}O_2$ max, although at submaximal work loads, heart rate and oxygen consumption were significantly lowered.

Keens et al. (1977) observed the influence of ventilatory muscle endurance training in four normal subjects. Four normals served as controls. The training consisted of sustaining maximal normocapnic hyperpnea 5 days a week for 4 weeks. Following the 6 week program of respiratory muscle training, the four normals showed a rise of 22% in the maximal level of ventilation capable of being sustained for 15 minutes under normocapnic conditions.

Effects of Ventilatory Muscle Training Programs on Diseased Individuals

Individuals suffering from bronchopulmonary diseases have benefitted from respiratory muscle training programs. Belman and Mittman (1980) discussed the effects of ventilatory muscle training upon selected respiratory functions of patients with chronic obstructive pulmonary disease. Following the protocol of Leith and Bradley (1976), each patient engaged in two 15-minute maximum sustained ventilatory capacity trials daily, attempting to reach a pre-set target. Post-test results revealed an increase of 31% in the maximum sustained ventilatory capacity. Submaximal endurance capacity for arm and leg exercise nearly doubled, and aerobic capacity, as measured by the distance covered in a 12-minute walk/run, increased 12%. Sergysels et al. (1979) likewise found significant elevations in ventilatory ability in response to a 6 month physical rehabilitation program which included breathing exercises.

Gilmenez et al. (1979) studied the effect of breathing maneuvers that were part of a 30-minute exercise program for 15 patients with chronic airway obstruction. The treatments occurred daily, with the length of the total program varying between 3 and 6 weeks. Results of the experiment showed an increase in the maximal mobility of the diaphragm, vital capacity, maximal flow at 50% of the vital capacity, and tidal volume at both rest and maximal exercise levels.

Anderson and his associates (1979) examined the influence of resistive breathing in 10 patients with severe chronic obstructive pulmonary disease. The patients trained for 30 minutes a day for 4 weeks. The procedure consisted of breathing against a resistive load that at times produced uncoordinated breaths. After 4 weeks, the load was raised and another 4-week session was initiated. As a consequence of the training, all of the individuals demonstrated that higher than normal resistances could be tolerated and sustained without undue fatigue. In addition, the subjects commented that the increase in respiratory muscle capability had enhanced their daily living activities, since they were now able to do more work without getting short of breath.

Keens et al. (1977) observed the effects of ventilatory muscle training on four adult subjects with cystic fibrosis. After determining the maximal sustainable ventilatory capacity (MSVC) that each subject could maintain for 15 minutes under normocapnic conditions, respiratory muscle training started. The program, which lasted 5 days a week for 4 weeks, consisted of each subject performing maximal normocapnic breathing to exhaustion for 25 minutes a day. After the training

period ended, the MSVC was remeasured. The results showed an average rise in the MSVC of 52%. In a similar study, Peress et al. (1979) found that a program of endurance breathing increased the MSVC by 23% and the MVV by 11%.

Effects of Respiratory Stress on Animals

Animal studies have revealed favorable adaptations resulting from respiratory stress techniques. Keens et al. (1978) produced a chronic respiratory load in rats by banding the extrathoracic trachea with arterial bonding tape. Five weeks after the initial banding, biochemical and histochemical analyses were performed on the diaphragm. The authors found that oxidative capacity, as indicated by succinate dehydrogenase activity, increased 38%, while the capacity for oxidation of fatty acids increased by 29%. Glycolytic activity was not affected. The intercostal muscles also showed similar alterations in aerobic metabolism.

Lieberman et al. (1972) studied the effect of treadmill exercise on guinea pigs. They found that daily endurance training enlarged the oxidative muscle fibers contained in the diaphragm, creating a larger oxidative capacity. Moore and Gollnick (1980, Abstract), examined the influence of an endurance treadmill running regimen on rats. They reported a two-fold increase in the succinate dehydrogenase activity of the diaphragm, leading to an enhanced endurance capability of the muscle.

Robertson et al. (1977) used an animal model to determine whether blood flow to the breathing muscles limited oxygen delivery and

work output during inspiratory resistance. They found an exponential rise in blood flow to the diaphragm; there were smaller increases to the other inspiratory and expiratory muscles. Based on blood lactate and pyruvate analyses, they concluded that no anaerobic activity was present.

In a later study, Robertson et al. (1977) studied the distribution of blood flow, oxygen consumption, and work output of the respiratory muscles during mechanical ventilation, resting spontaneous ventilation, and unobstructed hyperventilation facilitated by carbon dioxide rebreathing. In this animal model, flow to the diaphragm and external intercostals increased in the switch from mechanical to spontaneous ventilation. Furthermore, a continued linear ascent in blood flow occurred as ventilatory work was increased nine times by carbon dioxide rebreathing.

Effects of Non-Specific Training Programs on the Ventilatory Muscle Responses in Normal Individuals

The breathing musculature also responds to non-specific training methods. Shapiro et al. (1964), comparing athletes and non-athletes, noted that the presence of a "superior musculature" was a possible cause for the larger mean vital capacity and maximum breathing capacity of the athletes. Other researchers (Freedman et al., 1955; Stransky et al., 1979) have also found that the maximum breathing capacity was raised in subjects following a period of endurance training and muscular exercise.

Carey et al. (1956) studied diving instructors to ascertain whether their lung volumes increased in response to the stress of skin

diving. In a comparison between the instructors and other laboratory personnel, Carey showed that vital capacity, inspiratory reserve, and total lung capacity were significantly greater in the diving instructors. In a similar study, Crosbie et al. (1979) demonstrated that commercial divers were able to move large quantities of air in and out of their lungs in response to a progressive exercise load. This finding was attributed to the fact that the divers used exceptionally large tidal breaths and had reduced respiratory rates while working at elevated oxygen uptakes.

Milic-Emili et al. (1962) showed that at any given oxygen consumption, the pulmonary ventilation and the mechanical work of breathing were less in trained than in non-trained subjects. They concluded that "trained subjects might conceivably number among their endowments the ability to ventilate a large volume with a relatively small work requirement from their breathing muscles. Similarly, Claremont et al. (1977, Abstract) and Martin et al. (1979) have pointed out that endurance athletes seem to be endowed with low ventilatory responses, causing them to breathe less often than non-athletes at similar exercise levels. Martin et al. (1979) further suggested that low ventilation may be "the link between low ventilatory chemosensitivity and outstanding endurance athletic performance."

In a later study, Martin and Stager (1981) attempted to investigate a connection between athletic training of an endurance nature and ventilatory muscle endurance. Eight female endurance athletes and eight female non-athletes were compared in terms of short-term and long-term maximal ventilation. Both groups were paired according to age, body

size, and vital capacity. Martin and his associates reported that although the athletes and non-athletes had comparable short-term maximal ventilations (12-sec MVV), the athletes displayed much greater ventilatory endurance on two long-term breathing tests. The researchers attributed the differences in performance between the two groups to possible adaptations in the working skeletal muscles, such as increased mitochondrial density, myoglobin concentration, and tricarboxylic acid activity that would occur from endurance training.

Respiratory Muscle Fatigue as a Limiting Factor in Exercise

The next part of the discussion, investigating the role of respiratory muscle fatigue on exercise limitation, will be sub-divided into four sections: the development of a model of skeletal muscle fatigue; energy cost of breathing; efficiency of breathing; and muscle tension development.

A model of skeletal muscle fatigue. "Skeletal muscle fatigue (as opposed to central or neuromuscular transmission fatigue) occurs when the rate of energy consumption by a muscle is greater than the rate of energy supplied to the muscle by the blood" (Macklem and Roussos, 1977). Both Macklem et al. (1977) and Derenne et al. (1978) note that the muscle in this situation is forced to deplete its own energy supplies, and when they are exhausted, the muscle fails to produce power. Hence, the total energy consumption (total work performed by the muscle divided by its efficiency) of a fatigued muscle "is the sum of the energy stores plus the total amount of energy extracted from the blood by the muscle" (Macklem et al., 1977). If the

rate of oxygen delivery to the muscle is greater than or equal to the power requirement of the muscle, work can continue for an extended period of time. However, when the power requirement is larger than rate of oxygen delivery to the muscle, endurance time becomes fixed. Thus, in this model, a decrease in either efficiency or rate of energy supply will cause fatigue (Macklem et al., 1977).

An increase in muscle tension can also cause skeletal muscle fatigue. A muscle which contracts intermittently with equal contraction and relaxation will fatigue if a tension of 40% of more of maximum is developed (Roussos et al., 1976; Roussos et al., 1977). Furthermore, the percentage of maximal tension that a muscle is capable of developing has also been shown to influence the rate of energy consumption and fatigue (Roussos et al., 1979).

According to this model, a predisposition to muscle fatigue may depend upon factors involved in energy cost and supply, efficiency, and muscle tension development. The relationship of the fatigue components to the work of breathing constitutes the next part of this discussion.

Energy cost of breathing. The energy cost of breathing is a difficult measurement to make, due to differences and inadequacies among experimental methods (Otis, 1954; Robertson et al., 1977; Shepherd, 1966). Also, individual differences among subjects (Bradley and Leith, 1978; Martin and Stager, 1981; Milic-Emili and Petit, 1960) as well as variation in ventilatory depth and frequency (Cotes, 1963; Leith and Bradley, 1978; McGregor and Becklake, 1961; Otis, 1954) appear to further increase the variability of breathing cost. However,

some evidence is available estimating this measurement at different levels of ventilation.

Although the oxygen cost of breathing at rest is minimal, a commonly cited value is about 0.5 ml O_2/l of ventilation (Otis, 1954; Otis, 1964). During moderate exercise at a ventilation of about 30 liters per minute, the estimated oxygen cost of breathing varies from 1.2 to 2.1 ml O_2/l of ventilation (Campbell et al., 1959; Levison et al., 1968). At maximal levels of ventilatory work, there is considerable variability in the oxygen cost of breathing; values range from 2 to 9 ml O_2/l of ventilation (Bradley et al., 1978; Martin et al., 1981; McGregor et al., 1961; McKerrow and Otis, 1956; Shepherd, 1966). These data suggest that at high ventilations, the oxygen cost increases disproportionately such that "further increments of ventilation are ineffective in providing more oxygen for the body, because the oxygen cost of breathing becomes so great that all of the extra oxygen brought into the lungs by the extra ventilation is required by the muscles of respiration" (Tenney and Reese, 1968).

It seems, then, that an important condition restricting hyperventilation during vigorous exercise may be the oxygen cost of added respiratory effort. Research by a number of investigators verifies this assertion. Margaria et al. (1960) examined the work of breathing during muscular work at high ventilations. They found that the maximal useful pulmonary ventilation occurred when the energy cost of breathing in response to an increase in ventilation equaled the added energy provided by the same breathing increment. Any additional rise in ventilation, Margaria surmised, would not make extra energy available

for non-respiratory work, since the respiratory muscles would dominate any increase in oxygen supplied by an increase in ventilation. His data showed that the oxygen cost of breathing exceeded the added oxygen uptake at ventilations ranging from 120 to 170 liters per minute.

Shepherd (1966) reported that for ventilations between 90 and 130 liters per minute, the cost per minute was 4.4 ml O_2/l of ventilation at a controlled frequency of 50 breaths per minute and 4.3 ml O_2/l of ventilation at 100 breaths per minute. The useful ceiling of ventilation for these subjects was about 120 liters per minute BTPS, after which the oxygen cost of breathing would exceed the added oxygen intake. In another study, Shepherd (1967) measured the effects of exercise on the MVV and found that the useful ceiling of ventilation was approximately 135 liters per minute BTPS. Similar data cited by other investigators (Cotes, 1963; Otis et al., 1950; Tenney and Reese, 1968) shows that the oxygen cost of breathing is greater than the added oxygen supply at ventilation levels between 120 and 150 liters per minute.

McKerrow and Otis (1956) studied the oxygen cost of hyperventilation and concluded that the oxygen cost increased very rapidly with a small change in ventilation. The authors noted that this may have been due to either an increase in muscular movements not associated directly with respiration or to a greater drop in the mechanical efficiency of the breathing apparatus while sustaining high ventilations.

The high energy cost of breathing during heavy exercise may be affected by circulatory mechanisms. In a study by Ouellet et al. (1969), four subjects exercised at work loads ranging from submaximal to maximal. Values for heart rate, cardiac output, oxygen consumption, and pulmonary ventilation were reported. In three of the four subjects, an increase in oxygen consumption was witnessed after cardiac output had reached maximal values. In the fourth subject, maximal levels of cardiac output and oxygen consumption were achieved simultaneously. Using the data by Bartlett et al. (1958), Ouellet's group calculated the oxygen cost of breathing and the oxygen uptake minus the oxygen cost of breathing in one of his subjects. Their findings implied that the oxygen available to muscles not associated with respiration did not increase after circulatory parameters reached maximal limits. Rather, their data supported the conclusion by Riley (1960) that once a circulatory limit is established, "oxygen cost of breathing dominates the total cost of tissue maintenance" and "could reasonably be considered the limiting factor in exercise."

Efficiency of breathing. Wide disagreement exists in the literature concerning the efficiency of breathing. Values ranging from 1% to 25% have been reported (Bartlett et al., 1958; Campbell et al., 1957; Fritts et al., 1959; Margaria et al., 1960; Milic-Emili et al., 1960; Shepherd, 1966). Furthermore, Tenney and Reese (1968) have suggested that the higher efficiency calculations may be underestimated at high ventilatory rates.

A number of theories have been offered to reconcile these discrepancies. One explanation is that all the mechanical work

associated with breathing may not be directly measurable (Margaria et al., 1960; McGregor and Becklake, 1961; Otis, 1954; Robertson et al., 1977; Tenney and Reese, 1968). Another hypothesis is that the energy cost of isometric contractions necessary for postural maintenance may increase as breathing efforts become maximal (Otis, 1964). McKerrow and Otis (1956) state that this may "not be surprising when one sees how greatly people differ in the way they hyperventilate: some confine movement almost entirely to their respiratory muscles; others show large activity of the accessory musculature." In addition, the variance in rate and depth of breathing may also have an influence in the measurement of efficiency (Leith and Bradley, 1978; McGregor and Becklake, 1961; Milic-Emili and Petit, 1960; Otis, 1954).

Otis (1964) pointed out that the oxygen cost of breathing at low ventilations was a miniscule part of the total energy requirement; therefore, any inaccuracies in gas analysis or volume measurement could result in a gross error in oxygen cost which would affect the efficiency calculation. Efficiency is affected by differences in oxygen uptake determination (Robertson et al., 1977). Also, various means utilized in achieving extreme ventilation, such as voluntary hyperventilation, carbon dioxide rebreathing, and added dead space may alter the oxygen cost (Bartlett et al., 1958; McGregor and Becklake, 1961; Milic-Emili and Petit, 1960). In fact, evidence supports the argument that the oxygen cost of voluntary hyperventilation is greater than that of both resistance breathing and carbon dioxide rebreathing (Derenne et al., 1978; McGregor and Becklake, 1961; Otis, 1954).

Muscle tension development. Muscle tension development of the respiratory muscles is related to the degree of hyperinflation of the lung existing during exercise. As a result of the inherent force-length relationship of the breathing muscles, the higher the lung volume, the smaller the force generation and development for the same energy cost. Because of this, these muscles are more likely to suffer from fatigue at a high lung volume (Derenne et al., 1978). According to Roussos and Macklem (1977), hyperinflation also causes a "displacement of the respiratory system to a volume where the maximum inspiratory pressure the muscles can generate is diminished." If the diaphragmatic pressures exceed 40% of maximum, "the external power the respiratory muscles could produce without becoming fatigued would be reduced."

In an examination of diaphragmatic fatigue in man, Roussos and Macklem (1977) stated that respiratory muscle fatigue may limit exercise capacity in situations when a combination of hyperventilation and weak respiratory muscles are present. When this situation occurs, "the dynamic pressures generated are a greater fraction of the maximum possible than under normal circumstances, suggesting fatigue." An experiment demonstrating this pattern of fatigue was conducted by Roussos et al. (1979). He measured the time necessary to induce inspiratory muscle fatigue in five normal subjects who were breathing at functional residual capacity against a number of increasing resistive loads. In each test, a mouth pressure (Pm) was recorded that was a certain percentage of a maximum mouth pressure (Pmax). During the test, the five subjects were seated and were free to use their diaphragms or intercostal/accessory muscles, or both, to develop the

Pm. The data revealed that the Pm/Pmax ratio that could be sustained indefinitely was about 60%. As a result of these findings, Roussos and his associates suggested that "the rate of energy consumption is determined in large part by the tension the muscle develops relative to the maximum tension it is capable of developing."

Sharp et al. (1968) measured maximum inspiratory mouth pressures in individuals with chronic airway obstruction and concluded that "ineffectiveness or exhaustion of inspiratory muscles, working at disadvantageously short lengths . . . might be a major factor in the development of hypercapnia and that . . . failure of the inspiratory pump may be an important contributing factor in their ventilatory failure."

Exercise Ventilation

The effect of ventilation upon work limitation has been discounted by a number of investigators. The rationale presented in support of this position is that exercise ventilation at maximum aerobic capacity never results in a ventilation exceeding maximum breathing capacity; yet, hard, sustained work continues. Tenney and Reese (1968) found that the highest values achieved during exercise were in the range of 60% to 70% MVV, while Shepherd (1967) cited a figure of 70% to 80% MVV. These data, according to Freedman (1970), have encouraged investigators to interpret the difference between the 15-second MVV and the exercise ventilation as a "ventilatory reserve" which is presumably unavailable for use.

This conclusion, however, may not be a totally accurate one. A number of authors suggest that it may be unrealistic to relate sprint

ventilatory maneuvers like the MVV to exercise ventilations obtained at the end of maximal aerobic activity (Freedman, 1970; Hughes et al., 1968). In the opinion of Smidt (1979), to compare the MVV to exercise ventilation is "the same logic as to say that somebody can climb 10 stories with the same speed as one story." Freedman (1970) determined the maximal ventilation that a group of 20 subjects could maintain for 4 minutes during partial rebreathing. This figure was, on the average, 72% of the 15-second MVV. In eight of the subjects, additional values were also attained for ventilation during maximally tolerable steady-state exercise. The mean ventilation for these subjects was 80% of the 4 minute MVV, but considerable variability between subjects was evident. In four of the subjects, for example, exercise ventilation was 91% to 101% of the 4 minute MVV. Thus, these data suggest that exercise ventilation may reach maximal levels when compared to an appropriate endurance breathing measure. In addition, the ability to sustain maximal steady-state exercise may be related to the exercise ventilation.

In other research, Martin (1981) compared short- and long-term maximal ventilations of eight female endurance athletes and eight female non-athletes who were matched for age, body size, and vital capacity. The results of the experiment revealed that both groups had similar short-term maximal ventilations as determined by a 12-second MVV test. However, the athletes were superior to the non-athletes in two long-term breathing tests. In the first test, the athletes were able to reach a ventilation that was a significantly greater portion of their 12-second MVV measurement than did the

non-athletes. In the second test, 80% of the 12-second MVV was maintained by the athletes for 11 minutes as compared to 3 minutes for the non-athletes. Martin and his group postulated that the larger ventilatory capacities of the athletes may signify that ventilatory muscle fatigue occurs in exercise and "potentially influences exercise tolerance in normal persons."

Summary

The ability to sustain great breathing efforts, in both normal and diseased individuals, appears to be enhanced by a program of respiratory muscle training (Anderson et al., 1979; Belman and Mittman, 1980; Bradley and Leith, 1978; Gilmenez et al., 1979; Haas and Haas, 1981; Keens et al., 1977; Leith and Bradley, 1976). More general training techniques, non-specific to the ventilatory muscles, also appear to enlarge the capacity of these muscles to do more work (Carey et al., 1956; Crosbie et al., 1979; Freedman et al., 1955; Martin et al., 1979; Martin and Stager, 1981; Milic-Emili et al., 1962; Shapiro et al., 1964).

A possible etiology of respiratory muscle fatigue includes the rate of energy supply, the muscle efficiency, and the rate of muscle tension development (Derenne et al., 1978; Macklem and Roussos, 1977). Experimental evidence suggests that the high oxygen demand of the breathing muscles during maximal aerobic exercise may limit non-respiratory work (Margaria et al., 1960; McKerrow and Otis, 1956; Otis, 1954; Ouellet et al., 1969; Shepherd, 1966; Shepherd, 1967; Riley, 1960). Estimates of breathing efficiency differ widely in the

literature (Bartlett et al., 1958; Campbell et al., 1957; Fritts et al., 1959; Margaria et al., 1960; Milic-Emili and Petit, 1960; Shepherd, 1966) and appear to be related in part to differences in experimental methodology. In addition, the inability to further contract the respiratory muscles during high work loads and the functional disadvantage of operating at short, initial muscle lengths may also predispose these muscles to fatigue (Derenne et al., 1978; Roussos and Macklem, 1977; Sharp et al., 1968).

Finally, it appears that exercise ventilation during maximal steady-state work can be increased to very high levels through maximum work loads (Freedman, 1970) and respiratory muscle endurance training (Bradley and Leith, 1978; Leith and Bradley, 1976; Keens et al., 1977). Recent experiments (Martin et al., 1979; Martin and Stager, 1981) have further suggested that the ventilatory response during heavy, aerobic activity may be an important factor in exercise limitation.

CHAPTER III

METHODS

Subjects

Twelve subjects volunteered to participate in this study. The subjects were moderately active, but did not engage in any systematic endurance program, nor were any trained athletes. Of the 12, six were male and six were female. The 12 subjects agreed not to alter in any major way their mode of activity in regards to physical training. The experimental protocol was explained to each subject, and an informed consent form was signed by each subject prior to participation.

The true intent of the study was not disclosed to any of the subjects. Each participant was told that the primary focus of the investigation was to measure pulmonary function. The subjects were naive to the real purpose of the study; by doing this it was hoped that other means of obtaining an increase in maximal oxygen uptake would be kept to a minimum. The primary investigator administered two of the tests (the MVV test and the endurance breathing test), but was only peripherally involved in the maximal oxygen uptake test, acting as a spotter for the exercising subject. Trained laboratory personnel administered the maximal oxygen uptake test, following a protocol designed by the primary investigator. By implementing this procedure during the maximal oxygen uptake test, it was hoped that investigator

bias towards the experimental and control group would not be a factor in the experimental results.

To minimize possible psychological reactions to the novelty of the laboratory setting, each subject was exposed to experimental procedures and equipment similar to those of the actual test on one occasion prior to the first test day. Before the accommodation period, the subjects chose to participate in either the experimental or the control group.

Procedure

Ventilation tests. A 15-second maximum ventilatory volume (MVV) test was administered to each subject prior to the training period to determine maximum breathing capacity. Each subject breathed room air through a two-way Daniels valve connected to a rubber mouthpiece. One end of a Collins large spiral plastic tubing assembly (34 mm ID) was attached to the expired side of the Daniels valve. The other end of the tubing assembly was fitted with a two-way stopcock through which expired ventilation was collected. A meterological balloon was used to collect the expired gas. A noseclip was also used to prevent nasal breathing and to ensure total collection of the subject's ventilation. Once the experimental apparatus was securely in place, the subject assumed a standing position, and, at the command of the investigator, attempted to breathe maximally for 15 seconds.

The expired volume was measured by a Parkinson-Cowan CD-4 dry gas meter previously calibrated against a 120-liter Collins spirometer. Relative humidity, barometric pressure, and temperature were recorded

prior to each test, allowing conversion of the expired gas volume to BTPS conditions. Adequate rest time was allowed between tests to ensure recovery from hypocapnia. This procedure was repeated three times, with the highest value taken.

Following the 15-second MVV test, an endurance breathing test was performed by each subject. In this test, the subject attempted to ventilate at 100% of the previously determined MVV for as long as possible. The length of time that each subject was able to ventilate at this flow rate was recorded.

No encouragement was given to any of the subjects during either of the tests, and the subjects were unaware of their performance time on the endurance breathing task.

Maximal oxygen uptake test. A maximal oxygen uptake test was performed on each subject prior to the training period. The progressive running maximal test was conducted on a treadmill previously calibrated for both speed and grade.

Upon entering the laboratory on the test day, surface electrodes were affixed to the chest of the subject to allow monitoring of heart rate throughout the test. Once the electrodes were in place and the monitoring system was functioning, the subject sat in a chair and rested for 10 minutes. Next, the subject ran on the treadmill at a speed of either 170 (men) or 140 (women) meters per minute (m/min) and at a grade of 0% for 3 minutes.

Following this accommodation period, the subject stepped off to the side of the treadmill and rested for an additional 2 minutes.

After these preliminary procedures were completed, the maximal oxygen uptake test began. With the speed held constant at either 170 or 140 m/min, the subject started running at an initial grade of 0%. The grade was increased 2-1/2% every 2 minutes of the test until the subject was unable to continue.

Expired gas samples were collected at minute intervals during the last minutes of the test. The last four gas samples were analyzed with a Beckman LB2 analyzer for carbon dioxide and an Applied Electrochemistry Model S-3A analyzer for oxygen. The analyzers were calibrated with standardized gases previously measured by the Scholander method. Inspired ventilation was recorded with a Parkinson Cowan CD-4 dry gas meter fitted with a rotary potentiometer which allowed output to be channelled into a recorder (Narco Biosystems DMP-4B). A malfunction occurred in the gas meter halfway through the set of maximal oxygen uptake tests; this necessitated the measurement of expired ventilation. The expired volumes were collected in meteorological balloons which were then evacuated through a Parkinson Cowan CD-4 dry gas meter.

The oxygen uptake was calculated according to the formulas (Otis, 1964):

$$\dot{V}O_2 = \dot{V}_{I\ STPD} \left(F_{IO_2} - \frac{F_{IN_2}}{F_{EN_2}} \times F_{EO_2} \right)$$

$$\dot{V}O_2 = \dot{V}_{E\ STPD} \left(\frac{F_{EN_2}}{F_{IN_2}} \times F_{IO_2} - F_{EO_2} \right)$$

The carbon dioxide content of expired gas was calculated according to the formulas (Otis, 1964):

$$\dot{V}CO_2 = \dot{V}_{I\ STPD} \left(\frac{F_{IN_2}}{F_{EN_2}} \times F_{ECO_2} - F_{ICO_2} \right)$$

$$\dot{V}CO_2 = \dot{V}_{E\ STPD} \left(F_{ECO_2} - \frac{F_{EN_2}}{F_{IN_2}} \times F_{ICO_2} \right)$$

The usual criterion of a plateau or a drop in oxygen uptake in response to an additional work level was applied in determining the maximal oxygen uptake (Åstrand and Saltin, 1961). The highest value attained was used as the subject's maximal oxygen uptake.

Description of the training apparatus. A diagram of the apparatus is shown in Figure 1. An open circuit system was developed to allow the subjects to train at a specified percentage of their maximum breathing capacity. A variable output generator and a vacuum cleaner motor were screw mounted onto a wooden slab. A circular hole was cut out on the bottom of the slab directly underneath the motor to allow room air to enter the motor suction port. As room air entered the port, it was funnelled through the expiratory portion of the vacuum motor to a flow meter by a piece of Collins large spiral plastic tubing that was 34 mm ID. The air flow exited from the outlet side of the flow meter and entered the intake portion of a 120-liter Collins spirometer. Another tube connected the outlet portion of the spirometer to a three-way breathing valve, allowing continued air

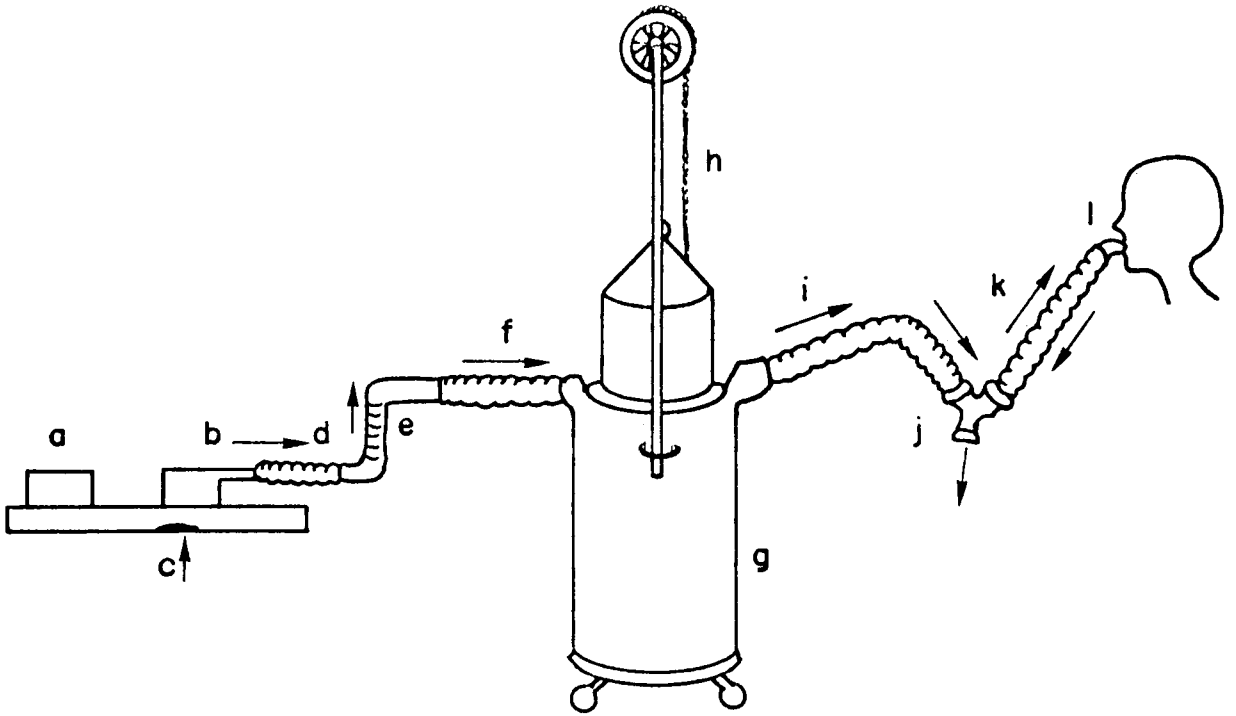

Figure 1. Diagram of the training apparatus used by the experimental group. The arrows indicate the direction of the air flow. Key: a = variable output generator, b = vacuum cleaner motor, c = air inlet, d = plastic tubing, e = flow meter, f = plastic tubing, g = spirometer, h = meter stick, i = plastic tubing, j = three-way valve, k = plastic tubing, l = rubber mouthpiece.

flow. Finally, a length of plastic tubing joined the three-way valve to a rubber mouthpiece from which the subject breathed.

Training protocol. On each training day, the subject assumed a position in front of the apparatus and placed the rubber mouthpiece into the mouth. A noseclip was used to prevent nasal breathing. After verification of subject readiness, the investigator activated the open circuit rebreathing system by switching the vacuum cleaner motor to the on position. This allowed 85% of the subject's MVV to enter the system. When the spirometer bell was approximately half full with room air, the investigator signalled to the subject to begin ventilation. The subject attempted to maintain the spirometer bell between two fixed marks 4 cm. apart on the meter stick affixed to the spirometer. The span between these two marks represented 5.3 liters. The subject performed this maneuver by ventilating at a rate equal to the flow rate into the spirometer. If the spirometer bell rose above the higher of the two fixed marks, the investigator instructed the subject to immediately return the spirometer to the training range. If the subject was unable to comply with this request, the apparatus was turned off and a training bout was completed.

Each subject engaged in four training bouts daily. The training bouts differed in time length from each other, with the first lasting 1 to 2 minutes, the second lasting 3 to 5 minutes, the third lasting 7 to 9 minutes, and the fourth lasting 10 to 12 minutes. A clock recorded the time achieved at the flow rate for each time interval. Adequate recovery periods followed each work bout. If the subject was

able to ventilate at the training intensity at each of the upper limits of the four time intervals, the training intensity was raised by 5% the next day. The subjects were encouraged to achieve maximum efforts each day and they were made aware of their own as well as other subjects' training records.

Once each week, the MVV was remeasured. If a new MVV figure was attained, a revised 85% training intensity was calculated and the subject worked at this level the following week. If 85% of the new MVV figure was not higher than the most recent daily training percentage, the latter was then used as the following week's initial training intensity. The endurance breathing test was also retaken. To standardize this measure, the subject always attempted to hold 100% of their original MVV value. The training period lasted from 45 minutes to 1 hour, for 5 consecutive days each week, for 5 weeks.

Post-Test Measurements

Pre-test measurements of height, weight, age, and body surface area were analyzed by use of a t-test for independent groups (Franks and Deutsch, 1973). Experimental results were analyzed by two methods. Covariance analysis (Bruning and Kintz, 1968) was used when the test for parallel slopes requirement (Neter and Wasserman, 1974) could be met. When the test for parallel slopes could not be met, a t-test for differences between groups on the difference between pre- and post-test values was performed. Both statistical procedures partialled out the effects of the pre-test scores and allowed adjusted post-test comparisons between the control and experimental groups to be made.

A $p<.05$ was used to ascertain whether differences between the groups on the post-test were caused by the experimental treatment. A Repeated Measures ANOVA (Bruning and Kintz, 1968) was performed on the experimental group data to test for significant differences. The Newman-Kuels post-hoc test (Sokal and Rohlf, 1969) was utilized to determine the location of the significant differences.

CHAPTER IV

RESULTS

The physical characteristics of the control and experimental groups are listed in Table 1. The two groups were similar in age, height, weight, and body surface area.

The experimental group showed significantly greater changes in the 15-second MVV than the control group (Figure 2). The mean post-test values for the experimental and control groups were 193 $\pm$ 13 and 155 $\pm$ 18 l/min, respectively. Figure 3 depicts the mean week-by-week changes in the MVV of the experimental group over the 5-week training program. The MVV values for weeks 1 through 5 were significantly greater than the MVV pre-test value. Furthermore, the value for week 5 was significantly greater than the value for week 1. The greatest change between two adjacent time periods occurred between the pre-test and week 1. The total increase in the 15-second MVV over the 5-week program was 28.6%.

The experimental group showed significantly greater changes in endurance breathing time than the control group (Figure 4). The mean post-test values for the experimental and control groups were 869 $\pm$ 30 and 31 $\pm$ 6 seconds, respectively. Figure 5 shows the mean week-by-week changes in the endurance breathing time of the experimental group over the 5-week training program. There were no significant differences in endurance breathing time between weeks 2 and 3, weeks 3 and 4, and

Table 1. Descriptive statistics of the experimental and control groups.

Group	Age (Years)	Height (cm)	Weight (kg) Pre	Weight (kg) Post	Body Surface Area (m^2)
Control (n = 6)	20 ± 1	170 ± 5	68.6 ± 6.7	69.4 ± 6.8	1.79 ± .10
Experimental (n = 6)	22 ± 2	174 ± 4	74.1 ± 2.7	74.3 ± 2.7	1.89 ± .03
p	p > .3	p > .5	p > .4	p > .5	p > .3

Values are $\bar{X}$ ± SE.

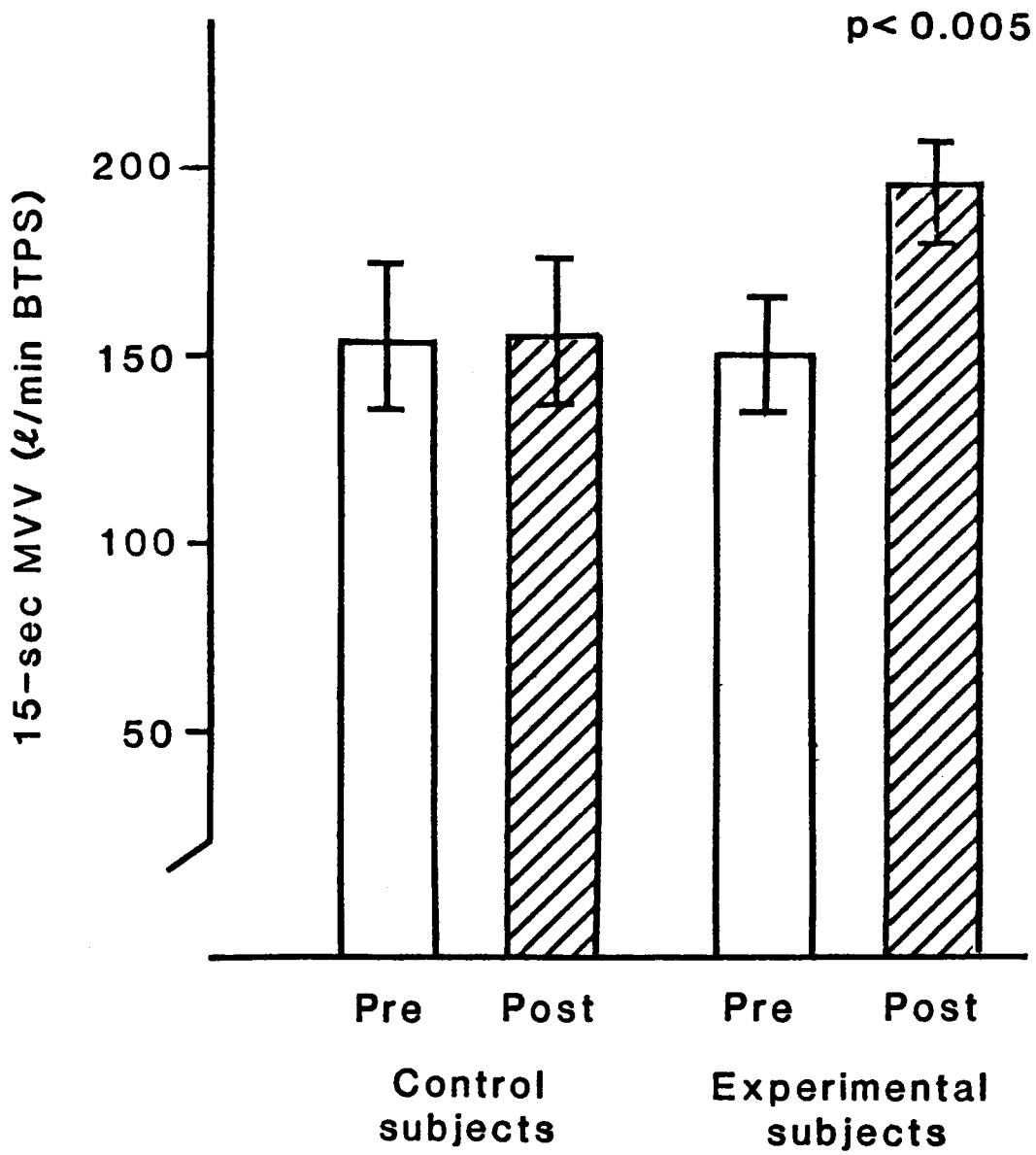

Figure 2. Pre- and post-test measurements of maximal ventilatory volume (MVV). Values are $\bar{X} \pm SE$. The adjusted post-test comparison between groups is significantly different ($p<0.005$).

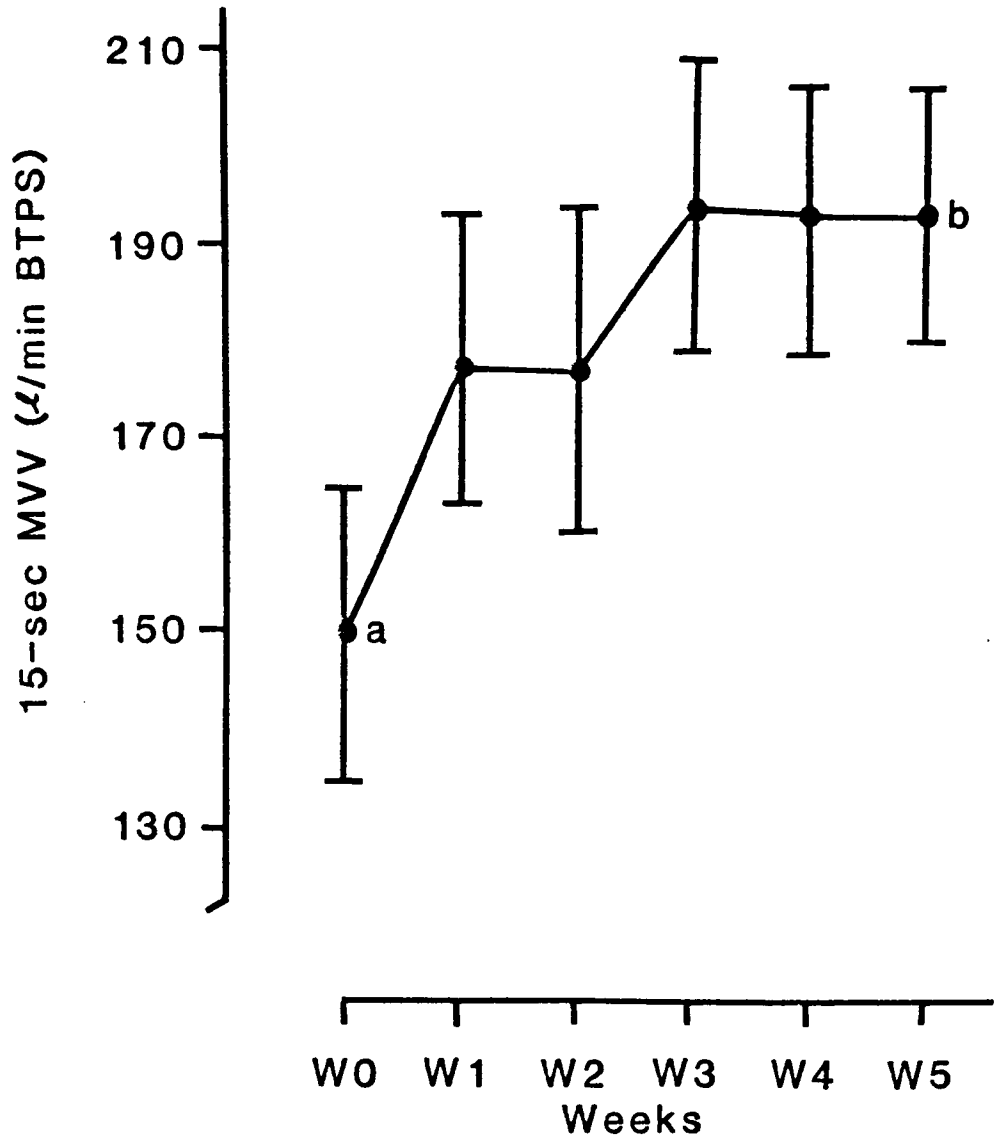

Figure 3. Week-by-week changes in MVV over the 5-week training program. Values are $\bar{X} \pm SE$. a = W0 is significantly different ($p<0.05$) from all other weeks; b = W5 is significantly different ($p<0.05$) from W1.

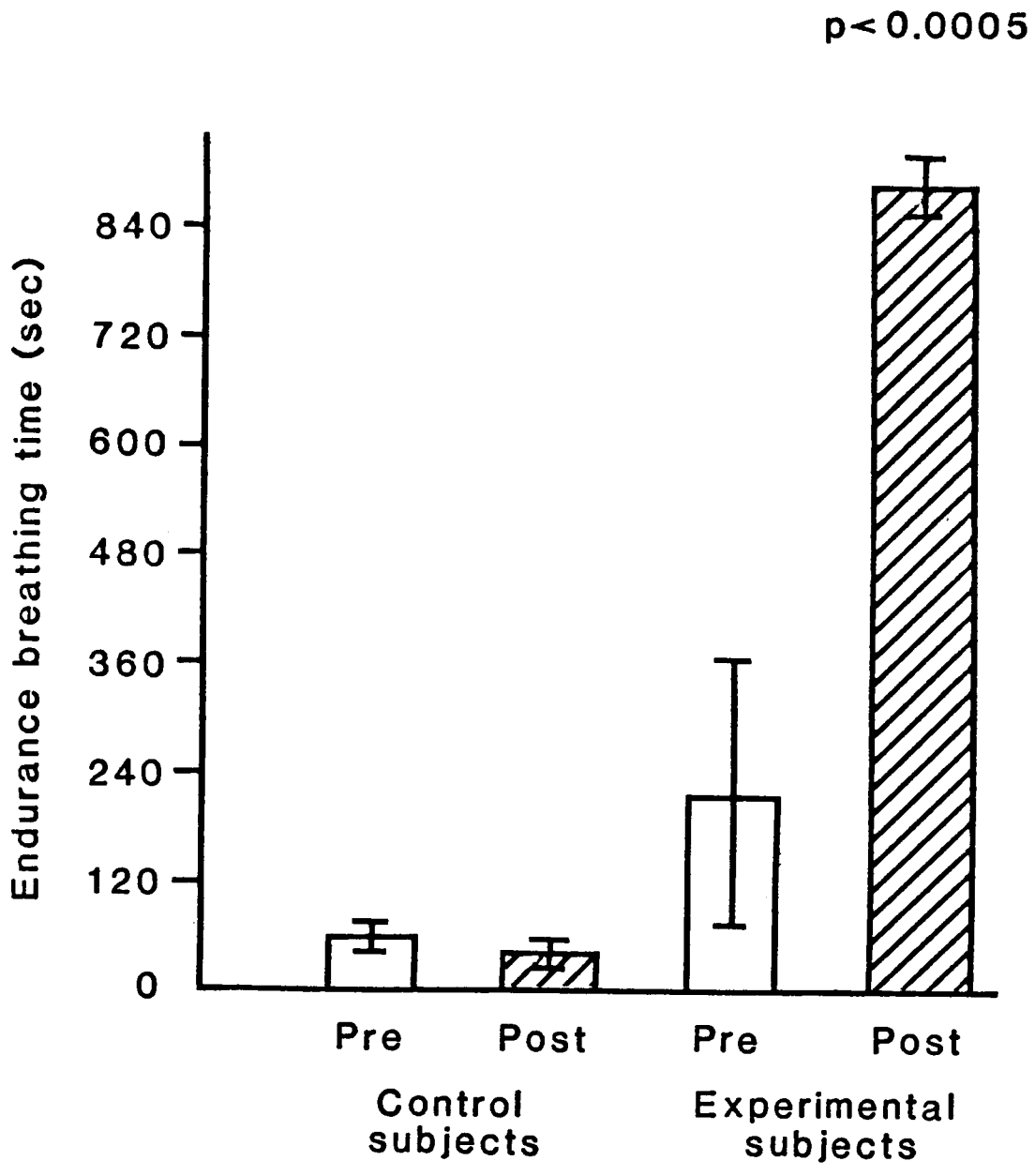

Figure 4. Pre- and post-test measurements of endurance breathing time. The endurance breathing test was terminated at 900 seconds. Values are $\bar{X} \pm SE$. The adjusted post-test comparison between groups is significantly different ($p<0.0005$).

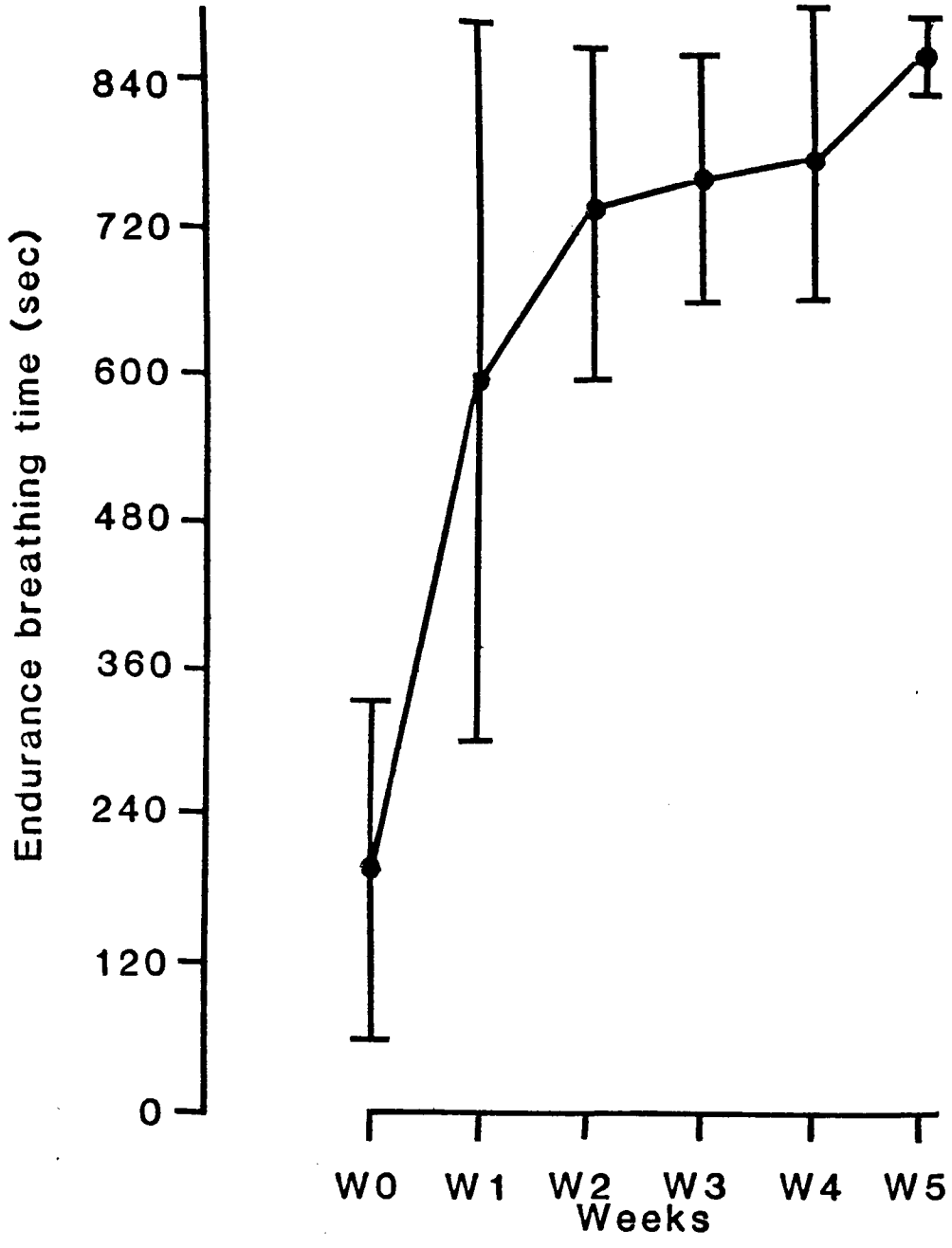

Figure 5. Week-by-week changes in endurance breathing time over the 5-week training program. The endurance breathing test was terminated at 900 seconds. Values are $\bar{X} \pm SE$. All weeks are significantly different ($p<0.05$) from each other except W2 vs. W3, W3 vs. W4, and W2 vs. W4.

weeks 2 and 4. All other weekly values were significantly different from each other. The greatest change between two adjacent time periods occurred between the pre-test and week 1. The total increase in the endurance breathing time over the 5 weeks amounted to 332%.

No significant changes were observed between the two groups in $\dot{V}O_2$ max (Figure 6); $.10>p>.05$. However, the experimental group made significantly greater changes in peak ventilation than the control group (Figure 7). The mean post-test values for the experimental and control groups were 133 ± 6 and 112 ± 14 l/min, respectively. The total increase in peak ventilation over the 5-week period was 13%.

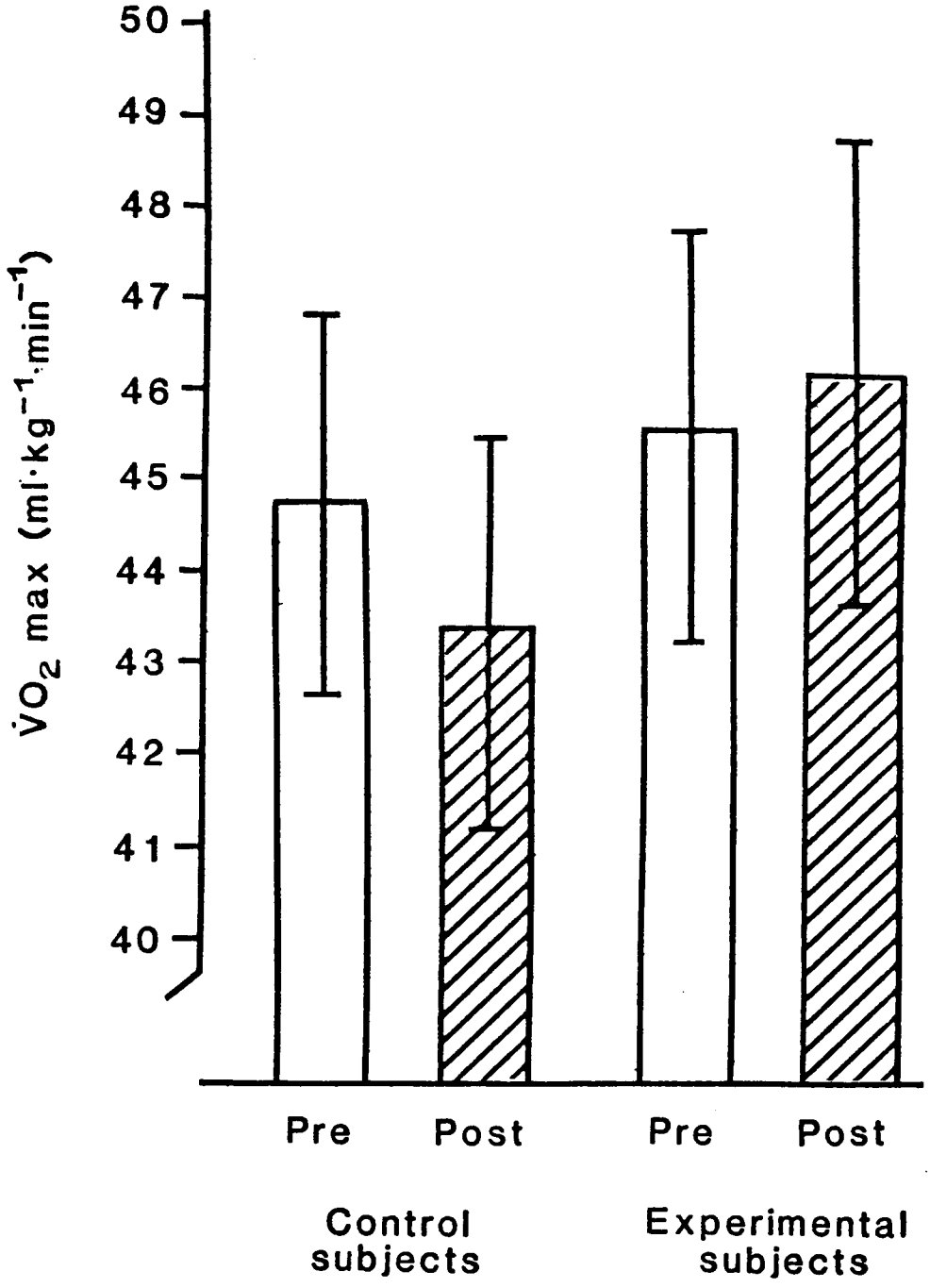

Figure 6. Pre- and post-test measurements of maximal oxygen uptake (VO_2 max). Values are $\bar{X} \pm SE$. The adjusted post-test comparison between groups is not significantly different (.10>p>.05).

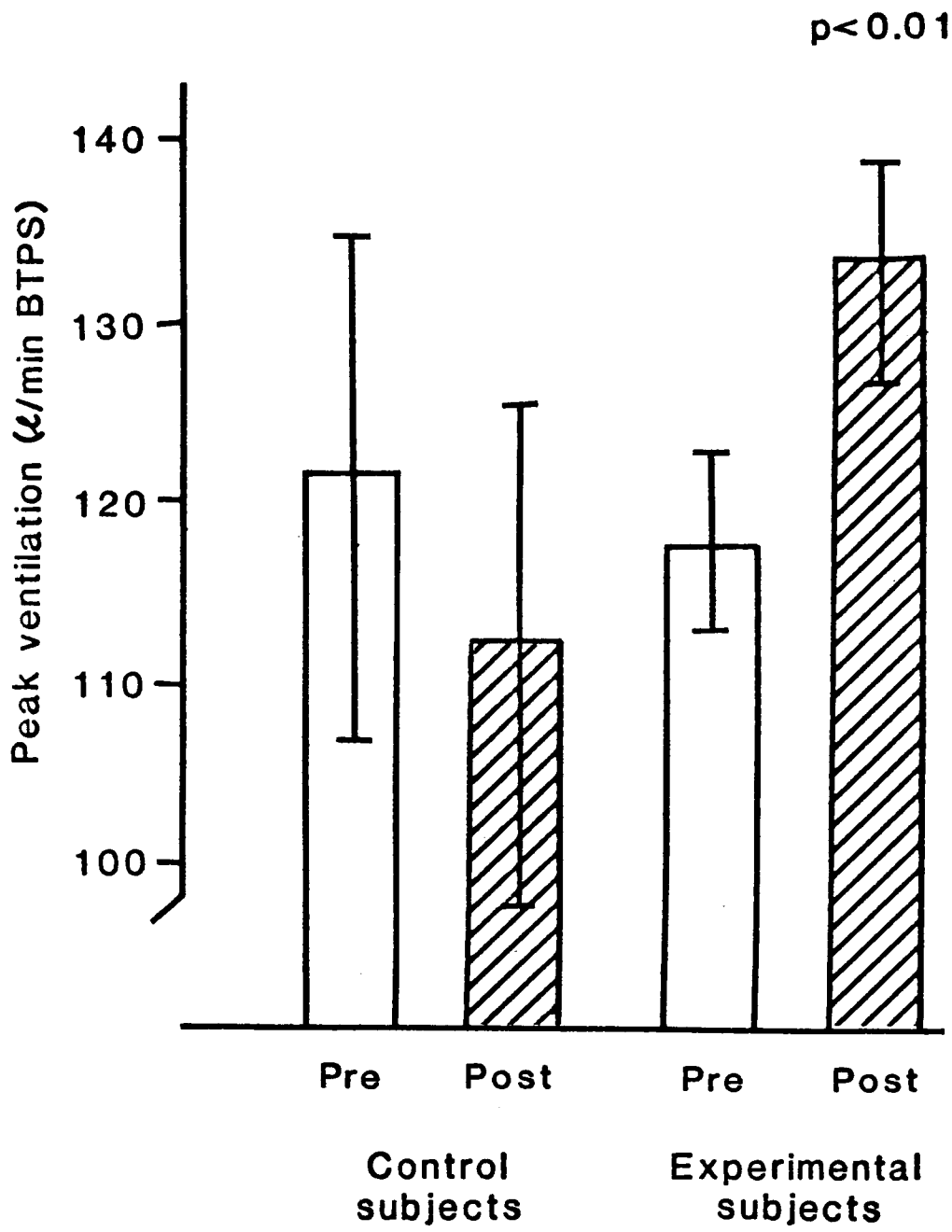

Figure 7. Pre- and post-test measurements of peak ventilation recorded during the $\dot{V}O_2$ max test. Values are $\bar{X} \pm SE$. The adjusted post-test comparison between groups is significantly different ($p<0.01$).

CHAPTER V

DISCUSSION

The 15-second MVV of the experimental group increased 29% over the 5-week training program. This ventilatory response was about two times as great as that recorded by Bradley and Leith (1976), who followed a similar protocol. The magnitude of the increase came as a surprise since the 15-second MVV maneuver is not a steady-state test; hence, it was not expected to respond in such a positive manner to endurance exercise. The intensity of the training program was quite high, and this may have been a powerful stimulus for the increase in the MVV. A comparison of the weekly training ventilation with the peak ventilation attained during the post $\dot{V}O_2$ max test is interesting in this respect. Appendix Table 4 shows that the mean training ventilations for weeks 2 (151), 3 (150), 4 (165), and 5 (164) were higher than the mean peak ventilations observed during the post $\dot{V}O_2$ max test (133). In addition, the training ventilation for week 1 (128) was only slightly less than the post-test peak ventilation. Thus, the subjects for the most part were training at a greater intensity than they would normally experience during maximal aerobic work.

Bradley and Leith (1976) have suggested that respiratory muscle endurance training may increase the 15-second MVV by altering the deleterious effects of hyperinflation. They surmised that "the

inspiratory muscles could now provide the necessary increased power, while operating at decreased lengths and increased velocities of shortening." In light of the role that hyperinflation plays in muscle fatigue (Derenne et al., 1978; Roussos et al., 1977; Sharp et al., 1968), it seems reasonable to suggest that the breathing program in the present experiment may have also been of sufficient intensity to counteract the adverse effects of hyperinflation, resulting in higher levels of MVV.

Most of the increase in the MVV (62%) occurred by the end of the first week. This indicates that a 5-week training period was not needed to produce significant changes. Recommendations for future research include a reduction in the length of the training period coupled with an increase in the sample size to provide greater statistical power.

The endurance breathing time of the experimental group increased 332% in 5 weeks. The magnitude of the increase would seem to indicate a substantial adaptation by the respiratory muscles to endurance exercise. Furthermore, the rise in endurance breathing time took place almost immediately. The effect of training on the level of maximum sustained hyperpnea was such that three of the six experimental subjects ventilated at 100% of their original MVV for 15 minutes after only 1 week of training. By the end of the second week, four subjects had reached this level, and by week 4, five of the six were maintaining their pre-test maximal ventilation for 15 minutes. These results are similar to those reported by Bradley and Leith (1976). They demonstrated that subjects who underwent respiratory muscle endurance

training were able to increase their level of sustained hyperpnea from 81% to 96% of their control MVV and raise their oxygen cost of breathing at this sustained hyperpnea by 67%. Their data were consistent with the idea that "a given ventilation required the same $\dot{V}O_2$ before and after training, but that high levels could be maintained much longer after training." The results from the present study also support work by Martin and Stager (1981) who showed that endurance trained athletes possessed higher peak $\dot{V}O_2$ during long-term MVV and had a higher endurance breathing capacity than did non-athletes.

It has been shown that strenuous endurance exercise causes an increase in the oxidative capacity of working skeletal muscle (Gollnick et al., 1972; Gollnick et al., 1973; Holloszy, 1967; Holloszy, 1975; Holloszy and Booth, 1976). These changes include increases in mitochondrial and capillary density, myoglobin concentration, and tricarboxylic acid cycle enzymes. The large overall increase in endurance breathing time suggests that such adaptations may have occurred in the respiratory muscles.

By the end of the first week of training, 59% of the total increase in endurance breathing time had been achieved. Also, the change in endurance breathing time by the end of the first week was almost identical to the change in the MVV (62%) during the same time period. This suggests that long-term and short-term maximal ventilation may be more related to each other than has been previously thought.

Respiratory muscle endurance training did not significantly increase the maximal aerobic power of the experimental subjects

(.10>p>.05). However, the experimental group showed a slight increase (1.0 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) in $\dot{V}O_2$ max while the control group showed a decrease (1.4 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$).

It is difficult to determine the cause for the nearly significant p value observed in $\dot{V}O_2$ max. A look at the individual scores of the experimental and control subjects (Table 2) reveals that the increases in endurance breathing time and 15-second MVV may have had only a marginal effect on $\dot{V}O_2$ max. Experimental subjects 2, 3, 4, and 5 registered increases across the board in endurance breathing time, MVV, and $\dot{V}O_2$ max; experimental subjects 1 and 6, however, showed increases in MVV and endurance breathing time but decreases in $\dot{V}O_2$ max. Addition of the control group data into this scheme further complicates the situation. Only two control subjects (5 and 6) reflected systematic changes in MVV, endurance breathing time, and $\dot{V}O_2$ max. The other four control subjects (1, 2, 3, and 4) did not register consistent differences across the three variables. Of particular note are the scores for subjects 3 and 4. Subject 3 showed the largest increase in MVV and the second largest increase in endurance breathing time, but registered the largest decrease in $\dot{V}O_2$ max. Subject 4, on the other hand, showed the second largest decrease in MVV and endurance breathing time, but recorded the largest increase in $\dot{V}O_2$ max.

It is also possible that the differences in $\dot{V}O_2$ max observed between the two groups simply reflected the decrease in $\dot{V}O_2$ max in the control group. Why did $\dot{V}O_2$ max drop off in the control group and not in the experimental group? Perhaps the experimental conditions between both groups were not the same. If this were the case, then one group

Table 2. Individual scores of the experimental and control subjects.

Group	MVV (l/min BTPS) Pre	MVV (l/min BTPS) Post	Δ MVV	End. Breathing Time (Seconds) Pre	End. Breathing Time (Seconds) Post	Δ EBT	$\dot{V}O_2$ Max ($ml \cdot kg^{-1} \cdot min^{-1}$) Pre	$\dot{V}O_2$ Max ($ml \cdot kg^{-1} \cdot min^{-1}$) Post	Δ $\dot{V}O_2$	Peak $\dot{V}_E$ (l/min BTPS) Pre	Peak $\dot{V}_E$ (l/min BTPS) Post	Δ Pk. V_E
CONTROL												
Subject 1	239	230	-9	9	17	+8	46.8	47.4	+.6	177	174	-3
Subject 2	156	157	+1	102	55	-47	46.9	45.7	-1.2	133	124	-9
Subject 3	159	176	+17	39	44	+5	52.9	48.8	-4.1	138	112	-26
Subject 4	125	121	-4	38	17	-21	40.0	40.7	+.7	92	82	-10
Subject 5	123	121	-2	37	25	-12	40.4	38.5	-1.9	95	97	+2
Subject 6	127	126	-1	42	29	-13	41.1	38.6	-2.5	93	85	-8
EXPERIMENTAL												
Subject 1	152	217	+65	141	900	+759	47.5	46.8	-.7	103	125	+22
Subject 2	199	242	+43	59	900	+841	51.1	51.9	+.8	142	157	+15
Subject 3	189	199	+10	32	718	+686	51.9	54.5	+2.6	114	132	+18
Subject 4	111	175	+64	900	900	0	40.9	43.2	+2.3	113	147	+34
Subject 5	124	163	+39	25	900	+875	39.9	41.3	+1.4	117	121	+4
Subject 6	124	159	+35	51	900	+849	41.6	39.4	-2.2	119	118	-1

might have been expected to perform differently regardless of the training stimulus. It is impossible to be certain that all influences were presented equally to both groups; however, a careful review revealed no striking dissimilarities between the two groups. Pre- and post-test measurements of maximal heart rate and weight in both groups showed no major differences. In addition, both groups appeared to be equally motivated. Temperature fluctuations between testing periods, however, were observed. The mean temperatures during the initial $\dot{V}O_2$ max tests were 22.4 and 22.6° C for the experimental and control groups, respectively. The post-test temperatures, though, were higher for both groups; the experimental group performed the test at a mean temperature of 25.3° C while the control group performed at 25.5° C.

The influence of this increase in mean ambient temperature on $\dot{V}O_2$ max was probably negligible. Nielsen and Nielsen (1962) have shown that environmental temperatures within the range of 5 to 30° C have little effect on body temperature rise in exercise. Other studies (Rowell et al., 1965; Rowell et al., 1966; Saltin et al., 1972; Williams et al., 1962) have also demonstrated no significant decrease in $\dot{V}O_2$ max in response to heat loads. It appears, then, that no adequate explanation can be found which properly addresses the question of the control group decrease in maximal oxygen uptake.

It is also possible that an increase in the sample size of the study might have raised the p value to a significant level. In any case, the preceding discussion illustrates both the ambiguity of the data and the need for cautious assessment in the explanation of changes in $\dot{V}O_2$ max associated with the present study.

The upward trend in $\dot{V}O_2$ max noted in this study may have reflected, in part, the high O_2 cost of severe ventilatory work. A number of investigators (Hughes et al., 1968; Otis, 1954; Otis et al., 1950; Margaria et al., 1960) have postulated that extreme ventilatory effort may limit oxygen delivery to non-breathing muscles. When this happens, a rise in oxygen uptake increasingly mirrors the oxygen cost of sustained hyperpnea. Shepherd (1966) and (1967) cited ventilations of 120 and 135 l/min BTPS as "break point" ventilations at which the oxygen cost of breathing exceeded added oxygen intake; others (Cotes, 1963; Tenney and Reese, 1968) have reported figures varying from 120 to 150 l/min. In the present study, the peak ventilation during the $\dot{V}O_2$ max test was 133 l/min BTPS, a ventilatory rate closely matching the aforementioned "break point" values.

No direct measurements of oxygen cost of breathing were made in this experiment. Thus, it is difficult to substantiate this explanation of $\dot{V}O_2$ max limitation. However, the oxygen cost of similar ventilatory work is available from sources and may be useful in evaluating the ventilatory mechanisms involved in maximal aerobic work.

Martin and Stager (1981) found that the oxygen cost of breathing during maximal ventilatory effort was about 9 ml O_2/l of ventilation. Multiplying this figure by the mean post-test peak ventilation of the control group yields an oxygen uptake of about 1.0 l/min for the oxygen cost of ventilation. The post-test control group mean $\dot{V}O_2$ max was 3.00 l/min. By dividing the oxygen cost of ventilation (1.0) by the $\dot{V}O_2$ max (3.00), the percent contribution of the oxygen consumption

of the breathing muscles to the maximal oxygen uptake can be obtained. This value for the control group is 33%.

Multiplying 9 ml O_2/l of ventilation by the post-test peak ventilation of the experimental group yields an oxygen cost of about 1.2 l/min of O_2 for the oxygen cost of ventilation. The mean oxygen uptake of the experimental group following training was 3.43 l/min. Therefore, the percent contribution of the oxygen cost of breathing to the total oxygen uptake (1.2/3.43) in this instance is 35%.

Using these data for base calculations, it is possible to show that an increase of about 200 ml O_2 (1200-1000) could have occurred as a result of respiratory muscle endurance training. This figure (200 ml O_2) expressed in terms of the range of subject weights seen in this study would be about 2.7 to 2.8 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$. The adjusted post-test increase in $\dot{V}O_2$ max between the two groups was 2.9 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$. Thus, it would seem that the oxygen cost of breathing data of Martin and Stager (1981) fit well with the experimental findings of this study.

However, if these data are correct, then close to one third of the total oxygen uptake is consumed by the respiratory muscles during maximal aerobic exercise. This value appears high when compared to those found in other studies. For instance, Nielsen (1936) estimated that the oxygen cost of breathing during high work loads was only 10% of the total body oxygen uptake. Margaria et al. (1960) showed that the relative energy expenditure of breathing during maximal work was an even lower percentage (3%) of the total energy uptake. Furthermore, since the mass of the respiratory muscles is small (Tenney and

Reese, 1968), it is unlikely that the energy production from these muscles can be very great. Thus, the oxygen cost of maximal ventilatory effort as determined by Martin and Stager (1981) may be too high a mean estimate for the work of breathing over the entire ventilatory range. However, it may be that 9 ml O_2/l of ventilation is an appropriate metabolic value for that part of the range involved in extreme ventilatory work, while a more conservative value may be proper for lower levels of ventilation. Further investigation into the relationship between $\dot{V}O_2$ max and respiratory muscle endurance training is clearly warranted, given the ambiguity of the results.

The peak ventilation sustained during the $\dot{V}O_2$ max test after training was significantly greater in the experimental group than in the control group. The importance of such a ventilatory adaptation may be in its ability to compensate for the metabolic acidosis present during heavy, aerobic work. Jones et al. (1977) found that the endurance time of subjects working at 95% of $\dot{V}O_2$ max was significantly reduced in an acidotic state (NH_4Cl capsule ingestion) and significantly increased in an alkolotic state ($NaHCO_3$ capsule ingestion). In addition, ventilation was highest in acidosis and lowest in alkolosis when the subjects worked at 66% $\dot{V}O_2$ max. If respiratory muscle fatigue occurs as a result of this heightened ventilatory effort, acid-base homeostasis may break down. However, if respiratory muscle endurance training may allow the ventilatory muscles to maintain acid-base equilibrium for a longer period of time through the continued excretion of CO_2. Such a respiratory compensation may lead to increased performance. Work by Martin et al. (1981) showed that

endurance athletes possessed longer endurance breathing times than non-athletes. Other studies have suggested that the ventilatory stress associated with endurance training may cause "low ventilatory chemo-responses" to sub-maximal metabolic work loads, thus reducing the fatigue component of the breathing muscles (Claremont et al., 1977, Abstract; Martin et al., 1979). A recent study by Sawka et al. (1980) also found that endurance trained athletes maintained a relatively constant ventilation during high intensity exercise. Taken together, these studies suggest that ventilatory muscle endurance may play an important role in acid-base balance during sub-maximal and maximal exercise.

An attempt was made in this experiment to determine the relationship of respiratory muscle endurance training and maximal aerobic performance. A running performance test designed to elicit subject fatigue in 3 to 8 minutes was included in the battery of pre- and post-tests. Due to temperature regulation problems in the laboratory, however, this test was eliminated. Nonetheless, respiratory muscle training may play a crucial role in maintaining acid-base balance during maximal aerobic exercise. This hypothesis appears worthy of further study and may prove useful in understanding the mechanisms which limit maximal performance.

BIBLIOGRAPHY

BIBLIOGRAPHY

Andersen, J. B., L. Dragsted, T. Kann, S. Johansen, H. Nielsen, K. Bay, E. Karbo, and L. Bentzen. Resistive breathing training in severe obstructive pulmonary disease. Scand. J. Resp. Dis. 60: 151-156, 1979.

Åstrand, P. O. and B. Saltin. Maximal oxygen uptake and heart rate in various types of muscular activity. J. Appl. Physiol. 16:977-981, 1961.

Asmussen, E. and M. Nielsen. Pulmonary ventilation and effect of oxygen breathing in heavy exercise. Acta Physiol. Scand. 43: 365-378, 1958.

Bartlett, R. G., H. F. Brubach, and H. Specht. Oxygen cost of breathing. J. Appl. Physiol. 12:413-424, 1958.

Belman, M. J. and C. Mittman. Ventilatory muscle training improves exercise capacity in chronic obstructive pulmonary disease patients. Am. Rev. Resp. Dis. 121:273-280, 1980.

Bradley, M. E. and D. Leith. Ventilatory muscle training and the oxygen cost of sustained hyperpnea. J. Appl. Physiol. 45:885-892, 1978.

Bruning, J. L. and B. L. Kintz. Computational Handbook of Statistics. Glenview, Ill.: Scott, Foreman, and Company, 1968, pp. 173-177, 222-225.

Campbell, E.J.M., E. K. Westlake, and R. M. Cherniack. Simple methods of estimating oxygen consumption and efficiency of the muscles of breathing. J. Appl. Physiol. 11:303-308, 1957.

Carey, C. R., K. E. Schaefer, and H. J. Alvis. Effect of skin-diving on lung volumes. J. Appl. Physiol. 8:519-523, 1956.

Claremont, A. D., J. A. Dempsey, P. G. Hanson, and W. G. Reddan. Ventilatory and acid-base responses to prolonged heavy work in endurance athletes (Abstract). Med. Sci. Sports 9:60, 1977.

Cotes, J. E. Exercise limitation in health and disease. Brit. Med. Bull. 19:31-36, 1963.

Crosbie, W. A., J. W. Reed, and M. C. Clarke. Functional characteristics of the large lungs found in commercial divers. J. Appl. Physiol. 46:639-645, 1979.

Demedts, M. and R. Anthonisen. Effects of increased external airway resistance during steady-state exercise. J. Appl. Physiol. 35:361-366, 1973.

Derenne, J.-Ph., P. T. Macklem, and C. Roussos. The respiratory muscles: mechanics, control, and pathophysiology: Part III. Am. Rev. Resp. Dis. 118:581-601, 1978.

Franks, B. Don and H. Deutsch. Evaluating Performance in Physical Education. New York: Academic Press, 1973, pp. 91-92.

Freedman, M. E., G. L. Snider, P. Brostoff, P. Kimelblot, and I. N. Katz. Effects of training on response of cardiac output to muscular exercise in athletes. J. Appl. Physiol. 8:377-47, 1955.

Freedman, S. Sustained maximum voluntary ventilation. Respir. Physiol. 8:230-244, 1970.

Fritts, H. W., J. Filler, A. P. Fishman, and A. Cournand. The efficiency of ventilation during voluntary hypercapnia studies in normal subjects and in dyspneic patients with either chronic pulmonary emphysema or obesity. J. Clin. Invest. 38:1339-1348, 1959.

Gilmenez, M., H. Uffoltz, G. Ferrar, P. Plouffe, and J. Lacoste. Exercise training with oxygen supply and directed breathing in patients with chronic airway obstruction. Respiration 37: 157-166, 1972.

Gollnick, P. D., R. B. Armstrong, B. Saltin, C. W. Saubert IV, W. L. Sembrowich, and R. E. Shepherd. Effects of training on enzyme activity and fiber composition of human skeletal muscle. J. Appl. Physiol. 34:107-111, 1973.

Gollnick, P. D., R. B. Armstrong, C. W. Saubert IV, K. Piehl, and B. Saltin. Enzyme activity and fiber composition in skeletal muscle of untrained and trained men. J. Appl. Physiol. 33: 312-319, 1972.

Haas, F. and A. Haas. Effect of inspiratory muscle training in healthy subjects (Abstract). Fed. Proc. 40:540, 1980.

Holloszy, J. O. Biochemical adaptations in muscle. J. Biol. Chem. 242:2278-2282, 1967.

Holloszy, J. O. Adaptation of skeletal muscle to endurance exercise. Med. Sci. Sports 7:155-164, 1975.

Holloszy, J. O. and F. W. Booth. Biochemical adaptations to endurance exercise in muscle. Ann. Rev. Physiol. 38:273-295, 1976.

Holmgren, A. and H. Linderholm. Oxygen and carbon dioxide tensions of aerterial blood during heavy and exhaustive exercise. Acta Physiol. Scand. 44:203-215, 1958.

Hughes, R., M. Clode, R. Edwards, H. T. Edwards, T. J. Goodwin, and N. L. Jones. Effect of inspired O_2 on cardiopulmonary and metabolic responses to exercise in man. J. Appl. Physiol. 24:336-347, 1968.

Jones, N. L., J. Sutton, R. Taylor, and C. Toewes. Effect of pH on cardiorespiratory and metabolic responses to exercise. J. Appl. Physiol. 43:959-964, 1977.

Keens, T. G., V. Chen, P. Patel, P. O'Brien, H. Levison, D. N. Crozier, and C. A. Bryan. Ventilatory muscle endurance training in normal subjects and patients with cystic fibrosis. Am. Rev. Resp. Dis. 116:853-860, 1977.

Keens, T. G., V. Chen, P. Patel, P. O'Brien, H. Levison, and C. D. Ianuzzo. Cellular adaptations of the ventilatory muscles to a chronic increased respiratory load. J. Appl. Physiol. 44: 905-908, 1978.

Knehr, C. A., D. B. Dill, and W. Neufeld. Training and its effects on man at rest and work. Amer. J. Physiol. 136:148-156, 1942.

Leith, D. E. and M. Bradley. Ventilatory muscle strength and endurance training. J. Appl. Physiol. 41:508-516, 1976.

Levison, H. and R. M. Cherniack. Ventilatory cost of exercise in chronic obstructive pulmonary disease. J. Appl. Physiol. 25: 21-27, 1968.

Lieberman, D. A., L. C. Maxwell, and J. A. Faulkner. Adaptation of guinea pig diaphragm muscle to aging and endurance training. Am. J. Physiol. 222:556-560, 1972.

Macklem, P. T. and C. S. Roussos. Respiratory muscle fatigue: a cause of respiratory failure? Clin. Sci. Mol. Med. 53:419-422, 1977.

Margaria, R., G. Milic-Emili, J. M. Petit, and G. Cavagna. Mechanical work of breathing during muscular exercise. J. Appl. Physiol. 15:354-358, 1960.

Martin, B. J., K. E. Sparks, C. W. Zwillich, and J. V. Weil. Low exercise ventilation in endurance athletes. Med. Sci. Sports 11:181-185, 1979.

Martin, B. J. and J. M. Stager. Ventilatory endurance in athletes and non-athletes. Med. Sci. Sports 13:21-26, 1981.

McGregor, M. and M. Becklake. The relationship of O_2 cost of breathing to respiratory mechanical work and respiratory force. J. Clin. Physiol. 40:971-980, 1961.

McKerrow, C. B. and A. B. Otis. Oxygen cost of hyperinflation. J. Appl. Physiol. 9:375-379, 1956.

Milic-Emili, G. and J. M. Petit. Mechanical efficiency of breathing. J. Appl. Physiol. 15:359-362, 1960.

Milic-Emili, G., J. M. Petit, and R. Deroanne. Mechanical work of breathing during exercise in trained and untrained subjects. J. Appl. Physiol. 17:43-46, 1962.

Moore, R. L. and P. D. Gollnick. Effect of endurance training on rat diaphragm (Abstract). Med. Sci. Sports 12:94, 1980.

Neter, J. and W. Wasserman. Applied Linear Statistical Models. Homewood, Ill.: Richard D. Irwin, Inc., 1974, pp. 703-707.

Nielsen, B. and M. Nielsen. Body temperature during work at different environmental temperatures. Acta Physiol. Scand. 56:120, 1962.

Nielsen, M. Die Respirationsarbeit bei Körperruhe und bei Muskel Arbeit. Skand. Arch. Physiol. 74:299-316, 1936.

Otis, A. B. The work of breathing. Physiol. Rev. 34:449-458, 1954.

Otis, A. B. The work of breathing. In: Handbook of Physiology, Sec. 3, Vol. II, W. Fenn and H. Rahn (Eds.). Washington, D.C.: American Physiological Society, 1964, pp. 463-476.

Otis, A. B. Quantitative relationships in steady-state gas exchange. In: Handbook of Physiology, Sec. 3, Vol. II, W. Fenn and H. Rahn (Eds.). Washington, D.C.: American Physiological Society, 1964, pp. 681-698.

Otis, A. B., W. O. Fenn, and H. Rahn. Mechanics of breathing in man. J. Appl. Physiol. 2:592-607, 1950.

Peress, L., P. McClean, C. R. Woolf, and N. Zamel. Ventilatory muscle training in obstructive lung disease. Bull. Europ. Physiopath. Resp. 15:91-101, 1979.

Riley, R. L. Pulmonary function in relation to exercise. In: Science and Medicine of Exercise and Sports, W. R. Johnson (Ed.). New York: Harper, 1960, pp. 162-77.

Robertson, C. H., G. Foster, and R. L. Johnson. The relationship of respiratory failure to the oxygen consumption of, lactate production by, and distribution of blood flow among respiratory muscles during increasing inspiratory resistance. J. Clin. Invest. 59:31-42, 1977.

Robertson, C. H., M. A. Pagel, and R. L. Johnson. The distribution of blood flow, O_2 consumption, and work output among the respiratory muscles during unobstructive hyperventilation. J. Clin. Invest. 59:43-50, 1977.

Robinson, S., H. T. Edwards, and D. B. Dill. New records in human power. Science 85:409, 1937.

Robinson, S. and P. M. Harmon. The effects of training and of gelatin upon certain factors which limit muscular work. Amer. J. Physiol. 133:161-169, 1941.

Roussos, C., M. Fixley, D. Gross, and P. Macklem. Respiratory muscle fatigue in man at FRC and higher lung volumes. Physiologist 19:370-374, 1980.

Roussos, C., M. Fixley, D. Gross, and P. T. Macklem. Fatigue of the inspiratory muscles and their synergic behavior. J. Appl. Physiol. 46:897-904, 1979.

Roussos, C. and P. T. Macklem. Diaphragmatic fatigue in man. J. Appl. Physiol. 43:189-197, 1977.

Rowell, L. B., J. R. Blackmon, R. H. Martin, J. A. Mazzarella, and R. A. Bruce. Hepatic clearance of indocyanine green in man under thermal and exercise stress. J. Appl. Physiol. 20: 384-394, 1965.

Rowell, L. B., H. J. Marx, R. A. Bruce, R. D. Conn, and F. Kusumi. Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. J. Clin. Invest. 45:1801-1816, 1966.

Saltin, B. and P. O. Åstrand. Maximal oxygen uptake in athletes. J. Appl. Physiol. 23:353-358, 1967.

Saltin, B., J. Blomquist, J. Mitchell, R. Johnson, K. Wildenthal, and C. Chapman. Response to exercise after bedrest and after training. Circulation 38:Suppl. 7, 1968.

Saltin, B., A. Gagge, V. Bergh, and J. Stolwijk. Body temperatures and sweating during exhaustive exercise. J. Appl. Physiol. 32: 635-643, 1972.

Sawka, M., R. Knowlton, and R. Glasser. Body temperature, respiration, and acid-base equilibrium during prolonged running. Med. Sci. Sports 12:370-374, 1980.

Sergysels, R., A. DeCoster, S. Degre, and H. Denolin. Functional evaluation of a physical rehabilitation program including breathing exercises and bicycle training in chronic obstructive lung disease. Respiration 38:105-111, 1979.

Shapiro, W., C. Johnston, R. Dameron, and J. Patterson. Maximum ventilatory performance and its limiting factors. J. Appl. Physiol. 19:199-203, 1964.

Sharp, J., P. Van Lith, C. Nuchprayoon, R. Briney, and F. Johnson. The thorax in chronic obstructive lung disease. Am. J. Med. 44:39-46, 1968.

Shepherd, R. J. The oxygen cost of breathing during vigorous exercise. Quart. J. Exptl. Physiol. 51:336-350, 1966.

Shepherd, R. J. The maximum sustained voluntary ventilation in exercise. Clin. Sci. 32:167-176, 1967.

Smidt, U. Respiratory muscle fatigue as limitation for exercise. Prog. Resp. Res. 11:215-223, 1979.

Sokal, R. R. and F. James Rohlf. Biometry. San Francisco: W. H. Freeman and Company, 1969, pp. 240-241.

Stenberg, J., B. Ekblom, and R. Messin. Hemodynamic response to work at simulated altitude, 4,000 m. J. Appl. Physiol. 21:1589-1594, 1966.

Stransky, A., R. Mickelson, C. Fleet, and R. Davis. Effects of a swimming training regimen on hematological, cardiorespiratory, and body composition changes in young females. J. Sports Med. 19:347-352, 1979.

Taylor, H. L., E. Buskirk, and A. Henschel. Maximal oxygen intake as an objective measure of cardiorespiratory performance. J. Appl. Physiol. 8:73-80, 1955.

Taylor, H. L. and L. B. Rowell. Exercise and metabolism. In: Science and Medicine of Exercise and Sport. W. R. Johnson and E. R. Buskirk (Eds.). New York: Harper and Row, 1974, p. 95.

Tenney, S. and R. Reese. The ability to sustain great breathing efforts. Resp. Physiol. 5:187-201, 1968.

Williams, C. G., G. Bredell, C. Wyndham, N. Strydom, J. Morrison, J. Peter, P. Fleming, and J. Ward. Circulatory and metabolic reactions to work in heat. J. Appl. Physiol. 17:625-638, 1962.

APPENDIX

Table 3. Pre- and post-test measurements of the dependent variables.

Group	MVV (l/min BTPS)		End. Breathing Time (Seconds)		$\dot{V}O_2$ max ($ml \cdot kg^{-1} \cdot min^{-1}$)		Peak $\dot{V}_E$ (l/min BTPS)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Control (n = 6)	154 ± 18	155 ± 18	45 ± 4	31 ± 6	44.7 ± 2.1	43.3 ± 2.1	121 ± 14	112 ± 14
Experimental (n = 6)	150 ± 15	193 ± 13*	201 ± 141	869 ± 30*	45.2 ± 2.2	46.2 ± 2.5	118 ± 5	133 ± 6*

Values are $\bar{X} \pm SE$.

*$p<0.05$.

Table 4. Week-by-week changes in MVV in response to respiratory muscle endurance training in the experimental group.

	MVV (l/min BTPS) Weeks											
Subject	0	Δ	1	Δ	2	Δ	3	Δ	4	Δ	5	Δ
1	152	-	177	+25	170	-7	194	+24	192	-2	217	+25
2	199	-	237	+38	256	+19	258	+2	252	-6	242	-10
3	189	-	199	+10	180	-19	208	+28	216	+8	200	-16
4	111	-	140	+29	138	-2	171	+33	173	+2	175	+2
5	124	-	164	+40	162	-2	182	+20	166	-16	163	-3
6	124	-	151	+27	159	+8	152	-7	161	+9	159	-2
$\overline{X}$	150	-	178	+28	177	-1	194	+17	193	-1	193	0
85% $\overline{X}$	128	-	151	+23	150	-1	165	+15	164	-1	164	0

Table 5. Week-by-week changes in endurance breathing time in response to respiratory muscle endurance training in the experimental group.

	Endurance Breathing Time (Sec) Weeks											
Subject	0	Δ	1	Δ	2	Δ	3	Δ	4	Δ	5	Δ
1	141	-	900	+759	900	0	900	0	900	0	900	0
2	59	-	548	+489	900	+352	683	-217	900	+217	900	0
3	32	-	59	+27	66	+7	304	+238	180	-124	718	+538
4	900	-	900	0	900	0	900	0	900	0	900	0
5	25	-	900	+875	900	0	900	0	900	0	900	0
6	51	-	283	+232	750	+467	900	+150	900	0	900	0
$\bar{X}$	201	-	596	+395	736	+140	764	+28	780	+16	869	+89

Table 6. Covariance analysis of maximal voluntary ventilation.

Source of Variation	Sums of Squares or Products								
	Y	X	XY	DF	Adj. SS	Adj. DF	Adj. MS	Adj. F	P
Treatments	4271.413	75.0	-565.992	1	5287.404	1	5287.404	22.14	p<.005
Error	14630.277	16538.85	14637.502	10	2149.052	9	238.784		
Total	18901.69	16613.85	13801.51	11	7436.456	10			

Table 7. Repeated measures ANOVA for the variable MVV.

Source	SS	DF	MS	F	p
Subjects	36560.533	5			
Treatments	8724.8667	5	1744.9733	13.65	p<.001
Error	3195.3003	25	127.812		
Total	48480.3	35			

Table 8. Newman-Kuels Post-hoc test for differences in MVV measured during the 5-week training program.

k	q (k, 25)	Rk ($q\sqrt{MSE/n}$)	Differences
6	4.36	20.226	W3 - W0 = 44*
5	4.16	19.298	W3 - W2 = 16 W4 - W0 = 43*
4	3.89	18.045	W3 - W1 = 16 W4 - W2 = 15 W5 - W0 = 43*
3	3.52	16.320	W3 - W5 = 1 W4 - W1 = 15 W5 - W2 = 15 W1 - W0 = 28*
2	2.91	13.490	W3 - W4 = 1 W4 - W5 = 0 W5 - W1 = 15* W1 - W2 = 0 W2 - W0 = 28*

Values are in l/min BTPS expressed as $\overline{X}$.

Ordered means: $\underline{W3}$ $\underline{W4}$ $\underline{W5}$ $\underline{W1}$ $\underline{W2}$ $\underline{W0}$
194 193 193 178 178 150

*p<.05.

Table 9. T-test for differences between the delta endurance breathing test values of the control and experimental groups following the 5-week training program.

Pre - Post	$(\text{Pre - Post})^2$	Pre - Post	$(\text{Pre - Post})^2$
+8	64	+759	576081
-47	2209	+841	707281
+5	25	+686	470596
-21	441	0	0
-12	144	+875	765625
-13	169	+849	720801
-80	3052	+4010	3240384

$\overline{X} = -13.33$ $\overline{X} = 668.3$

$SS_1 = 3052 - \frac{(80)^2}{6} = 1985.33$ $SS_2 = 3240384 - \frac{(4010)^2}{6} = 560367.3$

$$t = \frac{668.3 - (13.33)}{\frac{1985.33 + 560367.3}{10}} = \frac{681.630}{136.226} = 5.0036^*$$

*$p<.0005$.

Table 10. Repeated measures ANOVA for the variable endurance breathing time.

Source	SS	DF	MS	F	p
Subjects	1608840.8	5			
Treatments	1735302.5	5	347060.5	9.04	p<.001
Error	959844.7	25	38393.788		
Total	4303988	35			

Table 11. Newman-Kuels Post-hoc test for differences in endurance breathing time measured during the 5-week training program.

k	q (k, 25)	RK ($q\sqrt{MSE/n}$)	Differences
6	4.36	127.56	W5 - W0 = 668*
5	4.16	121.71	W5 - W1 = 273* W4 - W0 = 579*
4	3.89	113.81	W5 - W2 = 139* W4 - W1 = 184* W3 - W0 = 563*
3	3.52	102.98	W5 - W3 = 105* W4 - W2 = 44 W3 - W1 = 168* W2 - W0 = 535*.
2	2.91	85.14	W5 - W4 = 89* W4 - W3 = 16 W3 - W2 = 28 W2 - W1 = 140* W1 - W0 = 395*

Values are in seconds expressed as $\overline{X}$.

Ordered means: $\underline{W5}$ $\underline{W4}$ $\underline{W3}$ $\underline{W2}$ $\underline{W1}$ $\underline{W0}$
869 780 764 736 596 201

*p<.05.

Table 12. Covariance analysis of $\dot{V}O_2$ max.

Source of Variation	Sums of Squares or Products								
	Y	X	XY	DF	Adj. SS	Adj. DF	Adj. MS	Adj. F	p
Treatments	25.23	1.92	6.960	1	11.554	1	13.554	3.61	(.10>p>.05)
Error	285.697	274.077	262.7666	10	33.774	9	3.753		
Total	310.927	275.997	269.727	11	45.328	10			

Table 13. Covariance analysis of peak ventilation measured during the $\dot{V}O_2$ max test.

Source of Variation	Sums of Squares or Products								
	Y	X	XY	DF	Adj. SS	Adj. DF	Adj. MS	Adj. F	p
Treatments	1320.898	32.338	-206.688	1	1723.887	1	1723.887	13.33	p<.01
Error	6982.662	6764.562	6274.188	10	1163.299	9	129.255		
Total	8303.56	6796.9	6067.5	11	2887.185	10			

VITA

Don Whitford Morgan was born in Alexandria, Virginia on September 6, 1953. He attended elementary schools in Alexandria, Virginia and Honolulu, Hawaii. He graduated from The Kamehameha Schools in June, 1971.

In September, 1971, he entered Northwestern University, Evanston, Illinois. He received the Bachelor of Music Education degree in June, 1975.

In July, 1975, he accepted a position with Schilke Music Products, Chicago, Illinois, as a trumpet repairman. He left this position in June, 1979.

In September, 1979, he entered the Graduate School at The University of Tennessee, Knoxville. He received the Master of Science degree with a major in Physical Education in August, 1981. He has recently accepted the position of instructor in Physical Education at Andrews University, Berrien Springs, Michigan, as of July, 1981.

The author is a member of the American Alliance for Physical Education, Health, Recreation, and Dance. He is also a member of the American College of Sports Medicine.

The author is married to the former Deborah Leigh Hartsock of Hinsdale, Illinois.