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I am submitting herewith a thesis written by Luc E. Gosselin entitled "Effect of Ventilatory Muscle Endurance Training on Running Performance Time of Healthy Young Men." 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.

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EFFECT OF VENTILATORY MUSCLE ENDURANCE TRAINING ON RUNNING PERFORMANCE TIME OF HEALTHY YOUNG MEN

A Thesis

Presented for the

Master of Science

Degree

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ABSTRACT

The effect of ventilatory muscle fatigue on running endurance time on a treadmill was investigated. Five healthy young college males participated in the study. All subjects underwent both a control period and a ventilatory muscle endurance training period. Each period lasted three weeks. Subjects were measured for forced vital capacity (FVC), forced expiratory volume in one second (FEV), maximum breathing capacity (MBC), breathing endurance time, maximal oxygen uptake (max $\dot{V}O_2$), pre- and post-performance test lactate, peak exercise ventilation and peak $\dot{V}O_2$, and running endurance time on a treadmill. All measurements were recorded prior to a control period, after the control period, and after a three week ventilatory muscle endurance training program. Subjects underwent hyperpnea training (carbon dioxide rebreathing technique). The subjects ventilated at 85% of MBC for four different work bouts of varying duration (1-2 minutes, 3-5 minutes, 7-9 minutes, and 10-12 minutes). The subjects trained five days a week for three weeks. The one way ANOVA revealed that MBC, breathing endurance time, and running endurance time significantly changed during the study ($p < .05$). Further analysis revealed that running endurance time differed significantly between the pre- and post-control period rather than between the post-control and post-experimental periods. Based on these findings, it is concluded that ventilatory muscle fatigue does not limit running endurance time in fit young men.

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CHAPTER 1

INTRODUCTION AND REVIEW OF THE LITERATURE

Introduction

Åstrand and Rodahl list several variables that affect physical performance, including energy output, neuromuscular function, and various psychological factors. During intense exercise, energy output no doubt is of major importance. The ability to transform large quantities of chemical energy into mechanical work is influenced by a host of variables, some of which may be altered through training methods. In humans, the process of chemical breakdown and energy transfer can be accomplished either through anaerobic (without oxygen) or aerobic (with oxygen) pathways. For exercise of moderate to long-term duration, aerobic metabolism is of crucial importance.

Of the various factors that affect the limits of aerobic metabolism, the role of ventilation has received very little attention. Because arterial oxygen concentration and the diffusion of oxygen are essentially unaffected when fatigue occurs during intense exercise (Dempsey, 1977), ventilation is not thought of as a limiting factor of performance.

The Response of Ventilation to Exercise

In normal people, ventilation responds to exercise in an orderly and predictable manner. The response is brought about by control mechanisms tuned in with the metabolic demands of the body.

Thus, as the metabolic rate increases (as in exercise) ventilation also increases.

During light exercise, ventilation increases in proportion to work rate (Astrand and Rodahl, p. 251, 1977; Whipp et al., 1984). The linear increase occurs for both trained and untrained subjects. As exercise intensity increases, a point often referred to as the anaerobic threshold is reached. In trained individuals, the attainment of anaerobic threshold does not occur as quickly due to a greater capacity for aerobic metabolism (Bouhuys et al., 1966; Dempsey et al., 1977). Ventilation during work rates above anaerobic threshold increases out of proportion to increasing oxygen demand. The cause of the curvilinear increase in ventilation as the work rate intensifies has not been conclusively determined.

Several investigators suggest the curvilinear increase is a response brought about by the presence of lactic acid formed by anerobic metabolism of glycogen in the working muscles (Astrand and Rodahl, p. 251, 1977; Wasserman and Whipp, 1975; Jones et al., 1977). The respiratory system responds to the increased hydrogen concentration from lactic acid by increasing ventilation via central chemoreceptor stimulation (Jones et al., 1977; Bouhuys et al., 1966; Dempsey et al., 1977). This hypothesis would explain the delay of the curvilinear increase of ventilation in trained subjects because anaerobic threshold is not reached as quickly.

Although a correlation exists between the break in ventilation and the onset of blood lactic acid accumulation, a cause-effect

relationship has not been conclusively established. Hagberg et al. (1981), collected data on subjects suffering from McArdle disease. Because patients with McArdle disease lack the enzyme myophosphorylase, glycogenolysis is not possible, hence lactic acid cannot be formed. In the four subjects tested, ventilatory threshold occurred at 81% of $\dot{V}O_2$ max compared to 71% of $\dot{V}O_2$ max in the normal subjects. The ventilatory threshold occurred even in the absence of lactic acid with a resulting increase in venous pH due to a respiratory alkalosis and lack of a metabolic acidosis (lactic acid).

Despite inconclusive evidence on the cause of the break in ventilation, the response of ventilation to increasing work rates is clearly established. It seems possible that the response may be mediated by several factors, all of which may act by themselves or have synergistic effects upon one another.

Regardless of the stimuli, ventilation is greatly elevated during intense exercise and the ventilatory muscles are performing a substantial amount of work. Although peak exercise ventilation during intense exercise of short duration never equals maximum breathing capacity (MBC), it does achieve a considerable percentage of it. Tenney and Reese (1968) have found that peak exercise ventilation may reach 60-70% of MBC during intense exercise. The resulting increase in ventilation to this level no doubt stresses the respiratory muscles which may be subject to fatigue as other skeletal muscles are. It is, therefore, necessary to examine the amount of work performed by the respiratory muscles at rest and during exercise.

The Oxygen Cost of Breathing

The force generated by the respiratory muscles during rest--and the subsequent oxygen cost for the work performed--amounts to a very small fraction of the total oxygen uptake. Campbell et al. (1957) estimated that in normal, healthy individuals the oxygen cost per liter of ventilation is about 0.25 ml oxygen. Otis (1954) estimated this value to present only 1-2% of the total metabolic demand, whereas Margaria et al. (1960) estimated it to be less than 3%.

However, in certain disease states such as emphysema, the oxygen cost of breathing is greatly increased because of airway obstruction. The values reported for such individuals have varied and appear related to the amount of obstruction present (Otis, 1950). Campbell et al. (1957) reported values of 2-3 ml oxygen per liter of ventilation at a ventilation rate of only 10 l/min. At a ventilation rate of 20 l/min, the oxygen cost of breathing was as high as 7 ml oxygen per liter of ventilation. These values represent a much greater proportion of the total energy cost.

Several investigators (Milic-Emili et al., 1960; Otis, 1954; and Bartlett et al., 1958) have found that the oxygen cost of breathing increases as minute ventilation increases because of increased mechanical work performed by the respiratory muscles. The relationship is not a straight line, but one of continual increasing slope. Otis (1954) and Shepard (1966) have reported that the oxygen cost of breathing during intense exercise (a ventilation of 60-70% of MBC as reported by Tenney and Reese, 1968) may constitute

as much as 8-10% of the total metabolic demand. Shepard (1966) suggested that any increase in oxygen uptake above a ventilation of 120 l/min is used exclusively by the respiratory muscles.

Although athletes tend to ventilate less for a given workload than do the untrained individuals, there appears to be no difference in the work of breathing between the two groups for a given level of ventilation (Milic-Emili et al., 1962; Martin and Stager, 1981). Relative to maximal oxygen uptake, ventilation of both groups follows the same response (Åstrand and Rodahl, p. 230, 1977). Therefore, in spite of an athlete's greater aerobic capacity, both groups are ventilating large quantities of air during intense exercise--resulting in an increased work of breathing. It is apparent, then, that the respiratory muscles are doing a considerable amount of work during intense exercise and may be subject to fatigue as are other skeletal muscles. The result of respiratory muscle fatigue during intense exercise--should it occur--may have a significant effect on exercise performance.

Limiting Factors of Ventilation

Several studies have been conducted to determine the relationship between respiratory muscle work and respiratory muscle fatigue. Two different methods have been used to examine ventilatory muscle performance and include (1) the ability to sustain elevated levels of ventilation and (2) the ability to maintain a given fraction of

transdiaphragmatic pressure to maximal transdiaphragmatic pressure during inspiration--both for extended periods of time.

Maximum breathing capacity (MBC) refers to the maximum rate one can ventilate and is expressed as liters per minute. The term is misleading because the value is usually measured for only 10 to 15 seconds. In actuality, if one were to ventilate as rapidly as possible for 15 minutes or greater, the level of ventilation would decrease rapidly in the first minute and a half followed by a plateau which could be maintained for an extended period of time (Freedman, 1970; Tenney and Reese, 1968). The maximum amount of ventilation reported that could be maintained indefinitely was 50% of MBC by Freedman to 55% of MBC by Tenney and Reese.

Peak exercise ventilation during short-term intense exercise reaches a considerably higher value than 55% of MBC. Zocche et al. (1960) reported that only 53% of MBC could be maintained for 15 minutes or less, whereas Lieberman et al. (1973) reported a value of 82% of MBC for the same time period. Because higher values cannot be maintained, it seems reasonable to assume that respiratory muscle fatigue may occur if sufficient load is imposed upon them.

When one examines the relationship of the ability to maintain a given fraction of maximum transdiaphragmatic pressure during inspiration to the time it can be maintained, similar results are obtained. Roussos and Macklem (1977) revealed that the relationship between these two variables is curvilinear. When the fraction is high, fatigue occurs rapidly. However, as the fraction decreases

fatigue is delayed. The fraction of transdiaphragmatic pressure that can be maintained indefinitely is approximately 0.4. Therefore, like pulmonary ventilation, the induced inspiratory load (caused by the added resistance) applied to the respiratory muscles will cause respiratory muscle fatigue if the load is great enough.

Several variables influence the onset of fatigue, including fuel supply (Agostoni and Fenn, 1960), blood supply, and the type of exercise involved (Åstrand and Rodahl, p. 117, 1977). In part, exercise performance is dependent upon the histological and biochemical make-up of the muscle.

Lieberman et al. (1973) isolated guinea pig diaphragm and classified three types of muscle fibers. The three fibers consisted of a low oxidative-fast twitch muscle fiber, a high oxidative-fast twitch muscle fiber, and a high oxidative-slow twitch muscle fiber. These findings are consistent with those in other skeletal muscles (Holloszy and Booth, 1976). Lieberman et al. (1973) also found that 76% of the human diaphragm consisted of high oxidative-fast twitch and high oxidative-slow twitch muscle fibers. A clear relationship was established between the histochemical characteristics of the fibers and muscle performance. That is, the ability to maintain elevated levels of ventilation for prolonged periods of time is influenced by the muscle's capacity to provide energy through aerobic metabolism. Muscles that contain a majority of high oxidative fibers are therefore better equipped for aerobic metabolism.

It is apparent, therefore, that the ability to improve ventilatory endurance is linked to improving the oxidative capacity of

the muscles involved. Holloszy and Booth (1976) have noted that various training procedures can, in fact, cause certain histochemical changes to occur; the type of change dependent upon the training method utilized.

Trainability of Respiratory Muscles

In the past, two different techniques have been used to train the respiratory muscles. They include (1) hyperpnea training and (2) inspiratory loading. Although they differ from one another, they are both effective means to improve ventilatory endurance.

Hyperpnea training consists of having a subject breathe at a given target rate of MBC. The subject usually breathes through added deadspace tubing to maintain arterial blood PCO_2. Maintaining arterial PCO2 may also be accomplished by adding extra CO_2 on the inspiratory side. Hyperpnea training is often referred to as normocapnic hyperpnea. Following a few weeks of training via this method, several changes occur. Leith and Bradley (1976) reported a 14% increase in MBC. The increase appears to be due to an increase in the aerobic capacity of the respiratory muscles. In addition to an increase in MBC, ventilatory muscle endurance also increased. Bradley and Leith (1978) reported a 19% increase in the level of sustainable hyperpnea. This was accompanied by a 67% increase in oxygen consumption. Similar results were reported by Leith and Bradley (1976)--following endurance training the level of sustainable hyperpnea increased from 81% of MBC to 96% of MBC. Keens et al. (1977) reported that patients with cystic fibrosis increased ventilatory muscle

endurance by 51.6% whereas normal subjects increased it by 22.1%.

Respiratory muscle training by inspiratory loading has been used on patients with quadriplegia and patients with chronic obstructive pulmonary disease (COPD). Gross et al. (1980) reported that following eight weeks of training, patients with quadriplegia demonstrated improvements in both ventilatory muscle strength and endurance. This was indicated by a significant increase in peak negative inspiratory pressure (strength) and an increase in the critical resistance required to cause fatigue during the training sessions (endurance). In a similar study, Pardy at al. (1981) reported that patients with chronic airflow limitation increased ventilatory muscle endurance but had no increases in strength. No other changes in pulmonary functions were noted.

Training of the respiratory muscles through both methods caused a similar response--the muscles were able to maintain a greater load following training without fatigue occurring. The increase in oxygen consumption following hyperpnea training as reported by Leith (1976) strongly supports the idea that the increase in ventilatory muscle endurance is due to increased oxidative capacity of the respiratory muscles.

Keens et al. (1978) imposed inspiratory loading on rats by surgically narrowing their tracheas. Following five weeks of induced inspiratory loading through this method, the rats were sacrificed and the diaphragm was removed for analysis of oxidative, glycolytic, and beta-oxidative activity. Oxidative activity increased 38%, whereas beta-oxidative activity increased 29%. Gollnick et al. (1973)

reported that similar biochemical changes occur in other skeletal muscles following endurance training.

The increase in oxidative capacity of the respiratory muscles is accompanied by histological changes. Lieberman et al. (1972) reported that the percentage of red muscle fibers in guinea pig diaphragm increased 20% following training. However, Gollnick et al. (1973) reported that the size of red fibers and relative area they occupied increased following training. Thus, it appears that the increase in red fibers occurs in size rather than in the absolute numbers.

To this point it has been established that (1) the respiratory muscles are subject to fatigue providing the load is great enough and (2) ventilatory muscle endurance--through proper training techniques--can be improved (as a result of increased oxidative capacity of the muscle fibers). It has not been determined if respiratory muscles fatigue during severe, short-term exercise. If this is the case, then exercise performance could indeed be limited (or influenced) by the respiratory muscles. Two indirect studies have been conducted to determine if ventilation may limit performance.

Martin et al. (1982) induced respiratory muscle fatigue in subjects by having them ventilate as rapidly as possible (under normocapnic conditions for 150 minutes. During this time period, both ventilation and the subsequent oxygen cost of breathing decreased. Following this task, the subjects underwent a treadmill running test for endurance time. The subjects terminated the test sooner than they did on a similar test without having done previous ventilatory

work (6.5 minutes vs. 7.6 minutes, $p < .05$). The subjects also discontinued the test at a significantly lower heart rate, peak oxygen uptake, and peak ventilation. The significance of a reduced peak ventilation strongly supports the concept that respiratory muscle fatigue limited performance during the running treadmill test.

Dressendorfer et al. (1977) increased the work of breathing by adding resistance to inspiration during bicycle ergometer riding. As a result of the added inspiratory load, a significant reduction occurred in peak exercise ventilation and maximal oxygen uptake (max $\dot{V}O_2$) during the graded exercise test. Endurance time was also significantly less. When the added inspiratory load was removed at exhaustion, ventilation and max $\dot{V}O_2$ abruptly increased, allowing work to continue.

Based on this evidence, it seems reasonable to assume that respiratory muscle fatigue can limit exercise performance. If this assumption is true, then it would seem that improving ventilatory muscle endurance through training may have a positive effect on exercise performance.

Effect of Increased Ventilatory Muscle Endurance on Work Performance

Most of the work done investigating the effects of ventilatory muscle training on work performance has focused upon patients suffering from COPD. Pardy et al. (1981) reported that following a two-month training program of the respiratory loading, seven of twelve patients increased work endurance time on a cycle ergometer

greater than 40%. The same seven patients also increased maximal power output and peak oxygen uptake on a progressive exercise test. These changes were accompanied by an increase in ventilatory muscle endurance. In a less controlled study, Sonne and Davis (1982) obtained similar results.

Recently, Chen and Martin (1983) investigated the effects of improving ventilatory muscle endurance on work performance of nine healthy subjects. Following four weeks of training via inspiratory muscle loading, ventilatory muscle endurance significantly increased in the experimental group (the control group underwent no ventilatory muscle training). Additionally, the experimental group also had a significantly longer endurance time than the control group on a treadmill work performance test (435 sec. vs. 410 sec., $p < .05$). However, no changes were observed in forced vital capacity (FVC), forced expiratory volume in one second (FEV), 12 second MBC, ventilatory muscle strength, peak exercise ventilation, and max $\dot{V}O_2$ in either group. The results of this study support the concept that ventilatory muscle fatigue may affect performance of intense, short-term exercise.

Summary

The available literature pertaining to the role of ventilation during exercise at first glance appears confusing. Lack of any changes in arterial blood PO_2 and the diffusing capacity of oxygen during high rates of ventilation suggest that ventilation does not limit work performance. Additionally, even during intense work bouts

of short duration, peak exercise ventilation does not even approach MBC. This supports the concept of unused reserve of the respiratory system.

However, a great deal of literature exists revealing that ventilatory muscles are subject to fatigue--providing the work load is great enough. Furthermore, these muscles adapt to exercise as other skeletal muscles do and therefore can be trained. The response to training varies according to the type of technique used. Normocapnic hyperpnea generally alters two variables. First, MBC increases approximately 14% following training and second, ventilatory muscle endurance, as determined by the ability to sustain elevated levels of ventilation, also increases. Inspiratory loading, on the other hand, has no effect on MBC. Ventilatory muscle endurance (denoted by the ability to maintain a given fraction of transdiaphragmatic pressure to maximum transdiaphragmatic pressure) increases significantly following training. It has not yet been reported what effect inspiratory loading has on the ability to sustain elevated levels of ventilation.

Regardless of the method of training, increased ventilatory endurance appears to be caused by increased oxidative capacity of the respiratory muscles. In turn, it appears that improved ventilatory muscle endurance may improve work performance in subjects suffering from COPD and in normal, healthy subjects.

Statement of the Problem

Although Chen and Martin (1983) reported that ventilatory muscle endurance training appears to improve treadmill endurance time of normal individuals, the results are not yet conclusive because of some discrepencies in the study. These include (1) an absence of change in peak exercise ventilation and max $\dot{V}O_2$ in the experimental group despite increased endurance time, and (2) the unknown effect of inspiratory muscle loading on the ability of an individual to maintain elevated levels of ventilation.

It is the purpose of this study, therefore, to evaluate the effect of improving ventilatory muscle endurance (as determined by the ability to sustain elevated levels of ventilation) by normocapnic hyperpnea on treadmill running endurance of normal healthy subjects. It is hypothesized that improved ventilatory muscle endurance will significantly increase short-term, intense exercise performance.

CHAPTER 2

METHODS AND PROCEDURES

Introduction

Five healthy young men volunteered for the study. All were students enrolled at The University of Tennessee. Prior to any testing, an explanation of the study and procedures to be performed was given to each subject. An informed consent form approved by The University of Tennessee Committee on Research Participation was signed by each participant. The actual objective of the training program was withheld from each individual in order to minimize participant bias in the results. The subjects were familiarized with the equipment a day or two prior to any testing. They were also allowed to practice running on the treadmill and to practice the maneuvers in pulmonary function testing.

The study consisted of a control phase and an experimental phase, each phase lasting three weeks in duration. The subjects went through each one. The control phase was always the first. All measurements were made before the control period, after the control period, and after the experimental period.

Measurements

Pulmonary function measurements included forced vital capacity (FVC), forced expiratory volume in one second (FEV), 15 second MBC, and breathing endurance time. A 13 liter Collins spirometer was used to measure FVC and FEV.

The volume of expired gas for the fifteen second MBC maneuver was collected in a Douglas bag and later measured on a Parkinson-Cowan gas meter. While attached to a mouthpiece, the subject ventilated through 34 mm inner diameter (ID) deadspace tubing. The deadspace tubing was added to prevent hypocapnia (decreased arterial blood PCO2). The distal end of the deadspace tubing was connected to a low resistance valve (Koegel type). The expiratory port of the valve was connected to 34 mm ID tubing. A two-way valve was connected to the other end of this tubing. Expired gas was collected in a Douglas bag connected to the two-way valve.

FVC, FEV, and MBC were measured three times at each testing interval. The best value obtained during each testing interval was recorded.

Breathing endurance time was determined by having the subject ventilate at 100% of previously measured MBC for as long as possible. Flow was maintained at the predetermined rate by a vacuum cleaner motor (which in turn was controlled by a variable output generator). The motor pumped room air into a Tissot spirometer. The subject ventilated at a rate required to maintain the spirometer reservoir at a constant level (see Figure 1). When the subject was unable to continue to match the target ventilatory rate, the motor was turned off and the time was recorded.

Maximal oxygen uptake was determined by a graded exercise test on a treadmill. The speed was set at seven mph and the grade was elevated 2% every two minutes. Subjects ran to the point of exhaustion. Continuous encouragement was given throughout the test.

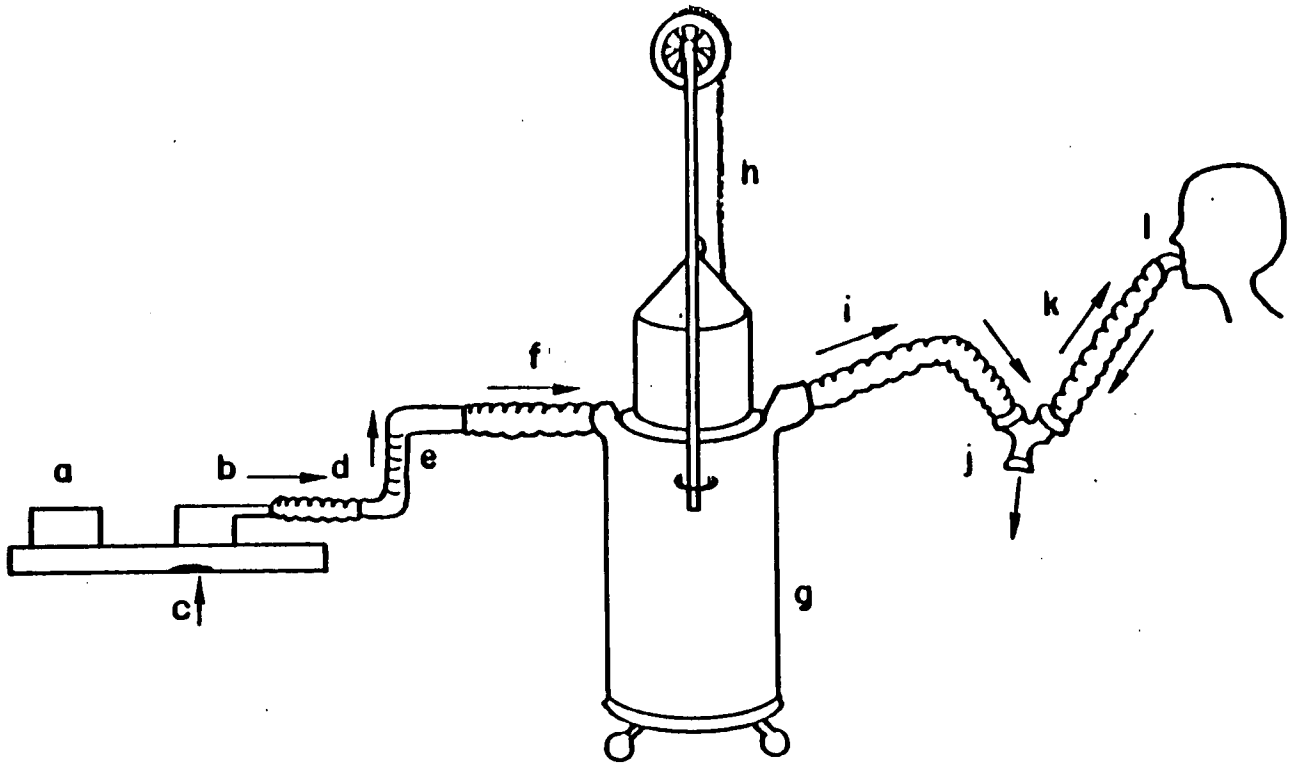

Figure 1. Diagram of the training apparatus used by the experimental group. The arrows indicate the direction of the air flow. Kay: a = variable output generator, b = vacuum cleaner motor, c = air inlet, d = plastic tubing, e = flow meter, f = plastic tubing, g = spirometer, h = meter stick, i = plastic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

Source: D. W. Morgan, Effects of respiratory muscle endurance training on maximal oxygen uptake in untrained men and women, Thesis, The University of Tennessee, Knoxville, 1981.

Inspired ventilation was measured by a Parkinson-Cowan gas meter connected to a four-channel recorder. The subject breathed through 34 mm ID low resistance tubing connected to a low resistance valve (Daniels type). Expired gas was collected in a Douglas bag for later analysis of gas fractions.

Expired oxygen and carbon dioxide were measured on an Applied Electrochemistry SA-3 and a Beckman LB-2 gas analyzer, respectively. Both gas analyzers were calibrated with known gas fractions from a cylinder analyzed by using the Scholander technique. Oxygen uptake was calculated using the following equation:

$$\dot{V}O_2 = VI\ (FIO2 - FIN2/FEN2 \times FEO2)$$

where $\dot{V}O_2$ is the volume of oxygen consumed, VI is the volume of inspired gas (STPD), FIO2 is the fraction of inspired oxygen, FEO2 is the fraction of expired oxygen, and FIN2 and FEN2 are, respectively, the fractions of inspired and expired nitrogen.

Following determination of maximal oxygen uptake via the graded exercise test, a full day of rest was given to each individual prior to the running endurance test. On the day of this test, the subjects were allowed to stretch and warm-up until they felt comfortable. The subjects then began running at seven mph on the treadmill. At this point, the treadmill was elevated to the grade required to elicit 100% of max $\dot{V}O_2$. When the treadmill reached the desired elevation, the clock was started. The subjects ran until they reached voluntary exhaustion. Oxygen uptake was calculated as previously described.

Verbal encouragement was not provided during the test. The subjects were made aware of this prior to the test. Additionally, the subjects were not permitted to know the outcome of the test. This was done as an attempt to prevent bias in the results by the subjects. The principal investigator of this study did not participate in the data collection of this test. Results of the endurance test were not known by the investigator until completion of the study. This was done as an attempt to prevent experimenter bias.

A blood sample was obtained from each participant during rest and four minutes after exhaustion by means of a finger stick. The blood was later used for measurement of lactic acid. An enzymatic technique designed for fluorometric analysis by Gutmann and Wahlfeld (1974) was used. All samples were analyzed in triplicate.

The ventilatory muscle training program was prescribed according to (1) intensity, (2) frequency, and (3) duration. Intensity was prescribed at 85% of MBC. The subjects trained for five days a week (frequency). The training program lasted three weeks (duration). The basis for the exercise prescription resulted from data reported in a previous study performed in this laboratory (Morgan, 1981).

During each training day, the subjects ventilated at 85% of MBC for four different work bouts with adequate rest periods between each work bout. The length of each work bout varied. The first work bout consisted of having the subject ventilate for a minimum of one minute and a maximum of two minutes. The second bout lasted from three to five minutes, the third lasted seven to nine minutes,

and the fourth lasted ten to twleve minutes. During each bout, the subjects were encouraged to ventilate as long as the minimum time indicated, and, if possible, for as long as the maximum time indicated. Flow was controlled by the same apparatus used for the breathing endurance test. If the subject fatigued and could not match the target rate equal that of the vacuum motor, the generator was turned off and the time was recorded. When the subject could maintain the desired flow for the full duration of each work bout, the flow was increased 5% the following day. The 15-second MBC was remeasured at the beginning of each new week of training. The training intensity was adjusted for any increase in MBC. The subjects were aware of their own progress as well as that of others. Following completion of the training program, all tests were repeated.

A one way analysis of variance (ANOVA) with repeated measures was used to analyze the data. A Post-Hoc Newman-Keuls multiple range test was used as the multiple comparison procedure for analysis of means when significance was established by the one way ANOVA.

CHAPTER 3

RESULTS

Table 1 contains the age and weight of the participating subjects. Mean weight remained constant throughout the duration of the study. The mean data for each testing interval are summarized in Table 2. Mean week by week changes in MBC are reported in Table 3. All other individual data for the three testing intervals are reported in tables appearing in the Appendix.

The one way ANOVA revealed that MBC, breathing endurance time, and running performance time had significantly changed during the study ($p < .05$). All other variables fell above the 5% confidence level. Further analysis of the means was done using a Newman-Keuls Post-Hoc test.

A significant difference for MBC existed between post-control and post-experimental measurements. There was no significant difference between pre-control and post-control values. Further analysis revealed that during training, week zero and week one were not significantly different from one another but both were significantly different from weeks two and three. Furthermore, week two and week three were not significantly different from one another (see Table 16, Appendix).

Post-experimental breathing endurance time was significantly greater than both pre- and post-control values. No significant difference existed between pre- and post-control values.

Table 1. Descriptive characteristics of the participating subjects.

Subject #	Age	Weight
1	22	63.0
2	25	84.4
3	20	83.9
4	21	81.9
5	22	67.8
Mean ± s.d.	22(1.8)	76.2(10.0)

Analysis of running endurance time revealed that the significant difference existed between the pre- and post-control values and not between post-control and post-experimental values.

Forced vital capacity and FEV did not change following training. These results agree with those reported by Leith and Bradley (1976), Morgan (1981), and Keens et al. (1977). MBC increased approximately 11% following three weeks of hyperpnea training. Though slightly less, this value is similar to those reported by Leith and Bradley (1976) and Morgan (1981). The smaller percent increase may be due to the shortened training program. In addition to the increase in MBC, ventilatory muscle endurance as determined by the ability to ventilate at 100% of pre-control MBC increased substantially. Only one of the five subjects was unable to ventilate at the given rate for ten minutes or more following training. For this reason, this particular subject may have benefitted from a fourth week of

Table 2. Summary data table of mean values at each testing interval (± s.d.).

Variable	Pre-control	Post-control	Post-experimental
FVC	5.8 (1.0)	5.6 (0.9)	5.6 (0.9)
FEV	4.6 (0.5)	4.5 (0.5)	4.6 (0.5)
MBC	212.2 (33)	212.5 (34)	235.1 (36)*
BET	38 (15)	39 (16)	540 (134)*
max $\dot{V}O_2$	53.6 (5)	52.2 (6)	54.0 (5)
Lactate			
pre-	1.2 (0.5)	1.0 (0.4)	1.5 (0.2)
post-	12.9 (1.8)	9.7 (2.8)	8.7 (1.6
Peak Ve	143.2 (20)	144.9 (19)	151.3 (18)
Peak $\dot{V}O_2$	---	52.9 (6)	53.8 (5)
RET	234 (78)	287 (92)	294 (104)*

*$p < .05$

FVC = forced vital capacity (1,BTPS)
FEV = forced expiratory volume (1,BTPS)
MBC = maximum breathing capacity (1/min,BTPS)
BET = breathing endurance time (seconds)
Peak Ve = peak exercise ventilation (1/min, BTPS)
RET = running endurance time (seconds)
max $\dot{V}O_2$ = maximal oxygen uptake ml O_2/kg per min)
Lactate = blood lactic acid (mM)

Table 3. Mean week by week changes in MBC during training.

Subject	Week 0	Week 1	Week 2	Week 3
Mean	213	216	228	235
+ s.d.	34	37	35	36

Values expressed 1/m (BTPS)

training. All other subjects were stopped after ten minutes as they all indicated that they could have continued for several more minutes. None of the five subjects was able to complete the first training bout at an intensity of 85% of MBC for ten minutes. The level of sustainable hyperpnea, therefore, increased approximately 15%. This value agrees with those reported by Leith and Bradley (1976), Bradley and Leith (1978), and Keens et al. (1977).

CHAPTER 4

DISCUSSION

In this study, running endurance time significantly increased but not as a result of improved ventilatory muscle endurance. A significant increase occurred between the pre- and post-control period. This increase occurred in the absence of an increase in max $\dot{V}O_2$ or an increase in ventilatory endurance. In spite of having the subjects practice treadmill running prior to the study (one practice run) it seems possible that a learning effect may be responsible for the increased running time. The absence of change in MBC, max $\dot{V}O_2$, or peak exercise ventilation reported by Chen and Martin (1983) suggests a similar learning phenomenon may be responsible for the reported increases in running time.

The lack of change in running endurance time following ventilatory muscle training suggests that ventilatory muscle fatigue does not play a role in limiting exercise performance--at least in healthy, fit individuals. However, subject three and subject five had an average max $\dot{V}O_2$ of less than 50 ml oxygen/kg per minute whereas subjects one, two, and four had an average max $\dot{V}O_2$ of about 57 ml oxygen/kg per minute. If these subjects are divided into two different groups, a different picture appears (see Figure 2 and Table 4). (Although the definitions of these terms are arbitrary, the two groups are labelled as fit (n=3) and unfit (n=2) for the purpose of discussion.) Although running endurance time increased in both

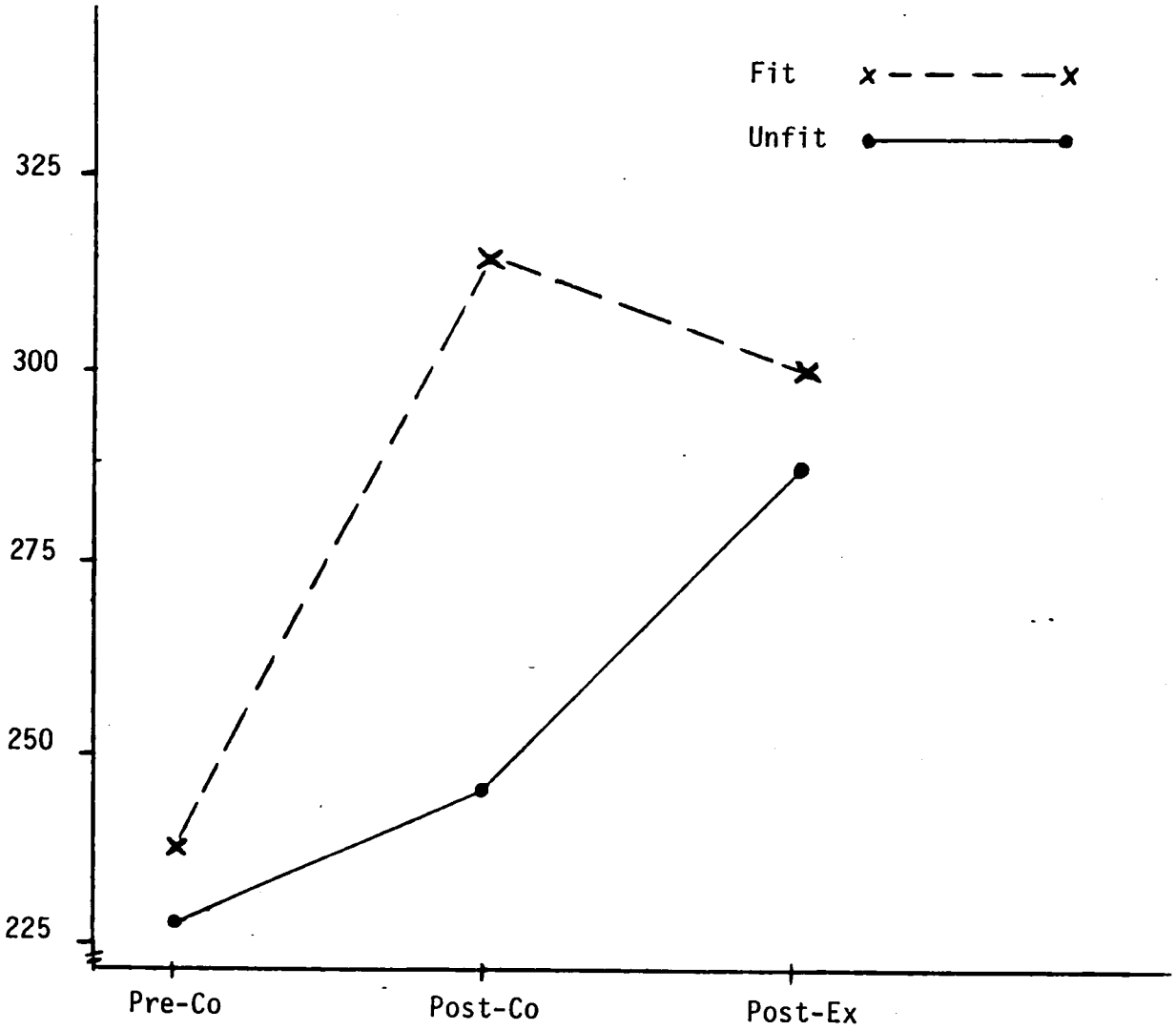

Figure 2. Changes in running endurance time at each testing interval between the fit and unfit subjects.

Table 4. Comparison of mean max $\dot{V}O_2$ and running endurance time between the fit and unfit subjects in the study.

	Mean max $\dot{V}O_2$			Mean RET		
Group	Pre-co	Post-co	Post-ex	Pre-co	Post-co	Post-ex
Fit	57.2	56.2	57.3	238	315	299
Unfit	48.3	46.3	49.0	228	246	288

max $\dot{V}O_2$ = ml oxygen/kg per minute (STPD)
Mean RET = mean running endurance time (seconds)
Pre-co = pre-control values
Post-co = post-control values
Post-ex = post-experimental values

groups, the increase was much more pronounced in the fit group following the control period. Following completion of ventilatory muscle training, the fit group demonstrated a decline in running endurance time. The unfit group, on the other hand, demonstrated more than a two-fold increase in running endurance time compared to their increase over the control period. This observation may warrant further research using unfit subjects.

Following three weeks of hyperpnea training, ventilatory muscle endurance (as measured by the ability to sustain elevated levels of ventilation for long periods of time) increased significantly in all the subjects. The increase in ventilatory endurance was probably a result of increased oxidative capacity by the respiratory muscles. Such an increase in oxidative capacity by the respiratory muscles is reflected by changes in the histochemical makeup of the individual muscle fibers.

Specific muscle fibers are better adapted to perform under anaerobic conditions whereas others are better suited for aerobic metabolism. Those that are better suited for aerobic metabolism are sometimes referred to as type I muscle fibers whereas those adapted for anaerobic metabolism are often referred to as type II muscle fibers (Astrand and Rodahl, 1977, p. 50). Of the type II muscle fibers, subgroups may exist. They are sometimes broken down into (1) type IIa (high oxidative potential, intermediate glycolytic capacity) and (2) type IIb (low oxidative potential, high glycolytic capacity) muscle fibers. A relationship appears to exist between physical performance and the histochemical makeup of the muscle fibers (Burke and Edgerton, 1975).

Because the energy requirement for a short, intense work bout (as in the 15 second MBC) is derived largely from anaerobic sources an increase in MBC would probably be due to an increased capacity for anaerobic energy production. Gollnick et al. (1973) demonstrated that following five months of endurance training at an intensity of 75-90% of max $\dot{V}O_2$, succinic dehydrogenase (SDH) activity and phosphofructokinase (PFK) activity increased 95% and 117% respectively in the exercising muscles. The increase in PFK activity (glycolysis) may have occurred in response to the intensity of the training program. In this study, all five subjects were ventilating at an intensity of at least 85% of MBC. If the subjects could complete the work bouts, the intensity was increased 5% the following day. This indeed occurred for all subjects during training. Thus, in addition to improving oxidative potential of the respiratory

muscles, it appears that glycolytic capacity may have also increased resulting in an increased MBC.

Chen and Martin (1983) demonstrated no such change in MBC during training via inspiratory loading. Keens et al. (1978) induced inspiratory loading in rats by surgically banding the trachea (causing an increase in airway resistance). Biochemical and histological analysis was performed on the diaphragm and intercostals five weeks post-surgery. Both beta-oxidation and oxidative activity increased significantly in both muscle groups yet no change occurred in PFK activity. It seems possible, therefore, that type IIa and type IIb muscle fibers are not recruited during inspiratory loading. If this is true, the need exists to determine the effects of inspiratory loading on the ability to sustain high levels of ventilation such as those encountered during intense exercise. Using inspiratory loading, improved ventilatory endurance has to this point always been denoted as the ability to sustain greater inspiratory loads and not as the ability to maintain a given fraction of MBC for an extended period of time (as in this study).

Another reason to question the results reported by Chen and Martin involves the role of exhalation during high levels of ventilation. While exhalation occurs passively during quiet breathing, expiratory muscles become actively involved under certain conditions--such as high rates of ventilation (Otis, 1954). To the author's knowledge, no studies have been conducted to determine the individual oxygen cost of exhalation vs. the oxygen cost of inhalation. If exhalation plays a significant role during intense

exercise, then inspiratory loading (which trains only the inspiratory muscles) would not seem to be the optimum means of improving total ventilatory muscle endurance.

Martin and Stager (1981) reported that in spite of athletes and non-athletes having similar 12 second MBCs, athletes possessed greater ventilatory endurance than the non-athletes. It appears that ventilatory muscle endurance obtained through normal training procedures by athletes is sufficient to meet the metabolic demands imposed by intense exercise and that ventilatory muscle fatigue does not play a role in limiting performance. Based on the report by Chen and Martin (1983), and of the results of this study, it appears that further work is needed to effectively determine if ventilatory muscle fatigue may limit performance in unfit subjects.

Recommendations for Further Studies

Future work on this topic should include several variations from the current experimental design. Care should be taken in order to obtain reliable test/retest results for the performance test. In order to minimize wide variations in the data, the following changes are recommended.

1. An extra practice run should be included at the beginning of the control period, with no test occurring sooner than one week apart.
2. In addition to the experimental group undergoing both the control and experimental period, a second group should be included to act as a control group. Such a group would

be tested at each testing interval but would not undergo any ventilatory muscle endurance training. Additionally, any subject who demonstrated a wide variation in performance test time over the control period should be dropped from the study. In order to accomplish this, the principal investigator should have access to the performance test results. However, in order to minimize experimenter bias, it is recommended that the investigator not participate in the data collection of this test.

3. The sample size should be increased from five to eight or ten subjects in each group.
4. Subjects should consist of those having max $\dot{V}O_2$'s of less than 45 ml oxygen/kg per minute.

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APPENDIX

Table 5. Individual pre-control data.

Subject #	FVC	FEV	MBC	BET	max$\dot{V}O_2$	RET	Lactate pre-	Lactate post-
1	4.72	3.79	168	32	57.9	232	1.4	15.4
2	6.77	4.87	225	53	58.9	134	1.8	12.5
3	4.95	4.31	198	16	50.4	203	---	----
4	6.77	5.10	255	51	54.8	348	0.8	11.0
5	5.54	4.74	215	36	46.2	252	0.8	12.6
Mean	5.75	4.56	212	38	53.6	235	1.2	12.9
± s.d.	0.98	0.52	33	15	5.3	78	0.5	1.8

FVC = forced vital capacity, liters (btps)
FEV = forced expired volume in one second, liters (btps)
MBC = maximum breathing capacity, liters/minute (btps)
BET = breathing endurance time, seconds
RET = running endurance time, seconds
max$\dot{V}O_2$ = maximal oxygen uptake, ml O2/kg per min (stpd)
Lactate= mmoles/liter

Table 6. Individual post-control data.

Subject #	FVC	FEV	MBC	BET	max$\dot{V}O_2$	RET	Lactate pre-	Lactate post-
1	4.71	3.58	162	46	55.9	246	0.6	12.1
2	6.59	4.77	228	45	57.5	249	1.3	6.3
3	5.00	4.42	206	17	46.1	228	---	----
4	6.58	4.99	255	57	55.3	450	1.4	11.9
5	5.15	4.55	212	30	46.4	264	0.8	8.6
Mean	5.61	4.46	212	39	52.2	287	1.0	9.7
± s.d.	0.91	0.54	34	16	5.5	92	0.4	2.8

Table 7. Individual post-experimental data.

Subject #	FVC	FEV	MBC	BET	max$\dot{V}O_2$	RET	Lactate pre-	Lactate post-
1	4.82	3.88	185	300	55.1	213	1.3	10.1
2	6.70	4.79	240	>600	58.0	227	1.3	9.8
3	4.97	4.45	237	>600	48.4	250	---	----
4	6.58	5.15	285	>600	58.9	457	1.7	6.7
5	5.06	4.50	229	>600	49.5	325	1.6	8.2
Mean	5.63	4.55	235	>540	54.0	294	1.5	8.7
± s.d.	0.93	0.47	36	134	4.8	101	0.2	1.6

Table 8. Individual performance test data at each testing interval.

Subject #	Peak Ventilation Pre-co	Peak Ventilation Post-co	Peak Ventilation Post-ex	Peak $\dot{V}O_2$ Pre-co	Peak $\dot{V}O_2$ Post-co	Peak $\dot{V}O_2$ Post-ex
1	121.3	130.7	128.3	56.1	54.8	54.9
2	168.3	163.5	163.8	60.3	57.5	54.9
3	154.0	149.9	158.1	----	48.7	46.7
4	149.4	161.0	170.0	55.3	58.6	39.7
5	123.2	119.6	136.4	45.3	45.1	51.1
Mean	143.2	144.9	151.3	----	52.9	53.8
± s.d.	20.4	19.2	18.0	----	5.8	5.0

Pre-co: pre-control values
Post-co: post-control values
Post-ex: post-experimental values
Peak Ventilation: l/min (BTPS)
Peak $\dot{V}O_2$: ml O2/kg per min (BTPS)

Table 9. Individual week by week changes in MBC during training. Values reported as liters per minute (btps).

Subject #	Week 0	Week 1	Week 2	Week 3
1	255	273	281	285
2	162	170	185	185
3	215	207	215	229
4	228	214	232	240
5	206	215	229	237
Mean	213	216	228	235
± s.d.	34	37	35	36

Table 10. ANOVA table for running endurance time.

Source	SS	DF	MS	F	P
Subjects	89844.266	4			
Treatment	10928.933	2	5464.466	4.993	.038
Error	8755.734	8	1094.467		
Total	109528.932	14			

Table 11. ANOVA table for breathing endurance time.

Source	SS	DF	MS	F	P
Subjects	24873.067	4			
Treatment	839014.535	2	419507.268	68.466	<.001
Error	49018.132	8	6127.266		
Total	912905.734	14			

Table 12. ANOVA table for MBC.

Source	SS	DF	MS	F	P
Subjects	13614.776	4			
Treatment	1731.449	2	865.724	22.358	<.001
Total	15655.996	14			

Table 13. Newman-Keuls Post-Hoc test for differences in breathing endurance time. Mean values reported as seconds.

Contrast	k	L	q(k,14)	Rk
3-1	3	502.4*	3.70	129.5
3-2	2	501.0*	3.03	106.1
2-1	2	1.4	3.03	106.1

M1	M2	M3
37.6	39.0	> 540.0

*(p< .05)
q = .95
n = 5

Table 14. Newman-Keuls Post-Hoc test for differences in running endurance time at each testing interval. Mean values reported in seconds.

Contrast	k	L	q(k,14)	Rk
3-1	3	60.6*	3.70	54.7
3-2	2	7.0	3.03	44.8
2-1	2	53.6*	3.03	44.8

M1	M2	M3
233.8	287.4	294.4

*(p< .05)
q = .95
n = 5

Table 15. Newman-Keuls Post-Hoc test for differences in MBC measured at the end of each testing interval. Mean values reported as 1/m (btps).

Contrast	k	L	q(k,14)	Rk
3-1	3	22.9*	3.70	10.3
3-2	2	22.6*	3.03	8.43
2-1	2	0.3	3.03	8.43

M1	M2	M3
212.2	212.5	235.1

*(p< .05)
q = .95
n = 5

Table 16. Newman-Keuls Post-Hoc test for differences in MBC during training. Mean values reported as 1/m (btps).

Contrast	k	L	q(k,14)	Rk
4-1	4	22.6*	4.11	11.44
4-2	3	19.1*	3.70	10.3
3-1	3	15.8*	3.70	10.3
4-3	2	6.8	3.03	8.43
3-2	2	12.3*	3.03	8.43
2-1	2	3.5	3.03	8.43

M1	M2	M3	M4
212.5	216.0	228.3	235.1

*(p< .05)
q = .95
n = 5

VITA

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Following training in respiratory therapy in Phoenix, Arizona, he began graduate school at The University of Tennessee-Knoxville in physical education. He received a Master of Science degree in March 1985. Future goals include further graduate work in the field of exercise physiology.