IMPROVED RESPIRATORY MUSCLE ENDURANCE OF HIGHLY TRAINED CYCLISTS AND THE EFFECTS ON MAXIMAL EXERCISE PERFORMANCE

by

MARY SUE FAIRBARN

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Department of Physical Education

The University of British Columbia
Vancouver, Canada

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ABSTRACT

To study the effects of 4 weeks of isocapnic hyperventilation training on the respiratory muscle (RM) endurance and cycling performance, 10 highly trained male cyclists ($\dot{V}O_2max$ = 66 ± 5 ml/kg/min) were assigned to equal experimental (E) and control (C) groups. The following measurements were obtained for each subject both the hyperpnea training period: maximal sustained ventilatory capacity (MSVC), maximal oxygen consumption ($\dot{V}O_2max$), maximal exercise ventilation ($\dot{V}_Emax$), a performance cycle test at 90% $\dot{V}O_2max$ (tlim), maximal ventilation during tlim ($\dot{V}_Etlim$), Forced expiratory volume in one second (FEV_1), Forced vital capacity (FVC), and Maximum voluntary ventilation in 12 seconds (MVV_{12}). For the E group, the training consisted of three, 8 minute intervals of hyperpnea per session, 4 times a week. Following training, the MSVC of the experimental subjects increased significantly (155.4 ± 11 to 173.9 ± 12 l/min; p = 0.004) with no change for the control group (155.1 ± 26 vs 149.5 ± 34 l/min, p > 0.05). $\dot{V}O_2max$ was not significantly changed for the E group (64.2 ± 1.9 vs 65.8 ± 4.8 ml/kg/min, p > 0.05) nor for the C group (68.0 ± 6.6 vs 67.1 ± 5.8 ml/kg/min, p > 0.05). Similarly, no significant differences were observed for tlim (342.2 ± 75 vs 427.8 ± 226.1 sec for the E group and 328.6 ± 99 vs 342.4 ± 80 sec for the C group, p > 0.05). There were also no significant changes for either

the E group or for the C group for the measurements of $\dot{V}_E$max (177.0 $\pm$ 22 vs 177.1 $\pm$ 13 l/min; 171.4 $\pm$ 36 vs 167.5 $\pm$ 21 l/min); $\dot{V}_E$tlim (176.0 $\pm$ 16 vs 178.5 $\pm$ 19 l/min; 174.0 $\pm$ 29 vs 176.3 $\pm$ 27 l/min); FEV_1 (4.4 $\pm$ 0.3 vs 4.5 $\pm$ 0.4 l/; 4.8 $\pm$ 0.6 vs 4.7 +. 0.7 l); FVC (5.5 $\pm$ 0.9 vs 5.7 $\pm$ 1.0 l; 5.7 $\pm$ 0.7 vs 5.6 $\pm$ 0.7 l); or MVV_{12} (205.5 $\pm$ 15 vs 216.3 $\pm$ 19 l/min; 215.2 $\pm$ 20 vs 223.3 $\pm$ 26 l/min, all $p > 0.05$). Results of this study indicate that the RM endurance of highly trained male cyclists can be increased following specific hyperpnea training but this does not result in changes in maximal exercise performance.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

List of Abbreviations

cmH_2O:	centimeters of water
CO_2 :	carbon dioxide
ECG :	electrocardiogram
EMG :	electromyogram
EELV:	end expiratory lung volume
$FECO_2$:	fractional mixed expired concentration of carbon dioxide
FEV_1:	forced expired volume in one second
$FICO_2$:	fractional inspired concentration of carbon dioxide
FRC:	Functional residual capacity
FVC:	Forced vital capacity
HeO_2:	Helium:oxygen
HR:	heart rate
IC:	Inspiratory capacity
J/min:	Joules per minute
l:	liter
MBC:	maximal breathing capacity
MEP:	maximal expiratory pressure
MIP:	maximal inspiratory pressure
ml/kg:	milliliters per kilogram
MSVC:	maximal sustainable ventilatory capacity

MVV_{xx}:	maximum voluntary ventilation for xx seconds
O_2:	Oxygen
PaO_2:	partial pressure of oxygen in arterial gas
PAO_2:	partial pressure of oxygen in alveolar gas
PCT:	performance cycle test
Pdi:	transdiaphragmatic pressure
Pes:	esophageal pressure
Pga:	gastric pressure
Pm:	mouth pressure
$\dot{Q}$:	cardiac output
RER:	respiratory exchange ratio
RM:	respiratory muscles
RR:	respiratory rate
SD:	standard deviation
SM:	sternomastoid
SVC:	sustainable ventilatory capacity
tlim:	time limit
$\dot{V}_A$:	alveolar ventilation
VC:	Vital capacity
VT:	tidal volume
$\dot{V}_E$:	ventilation
$\dot{V}O_2max$:	maximal oxygen consumption

$\dot{V}O_2$resp:	Oxygen consumption of the respiratory system
$\dot{V}$-V:	flow-volume
$\dot{W}$max:	Maximal work

INTRODUCTION

Traditionally, exercise scientists have not considered the ventilatory system to be a limiting factor to aerobic performance at sea level. Brooks and Fahey (5) review four arguments to support this belief. First, the alveolar partial pressure (PAO_2) increases during intense exercise and the arterial partial pressure of oxygen (PaO_2) remains constant; thus ventilation appears adequate to maintain PaO_2. Second, the ventilation/perfusion ratio ($\dot{V}_A/\dot{Q}$) as well as the ventilatory equivalent for oxygen ($\dot{V}_A/\dot{V}O_2$) both increase during exercise. Third, the alveolar surface area available for $\dot{V}_A$ is extremely large (50 m^2) compared to the capillary blood volume. Finally, the maximum $\dot{V}_E$ during exercise ($\dot{V}_E max$) does not reach the maximum voluntary ventilation (MVV) and $\dot{V}_E$ can be increased beyond $\dot{V}_E max$ volitionally. Therefore, it would appear that the ability of the ventilatory system to cope with the increased demand for oxygen during intense activity, exceeds similar abilities of the cardiovascular or metabolic systems.

There are, however, studies that report arterial oxygen desaturation in highly trained athletes during heavy work (12,16,34) which raises the possibility that O_2 delivery to muscles may contribute to limitation of peak performance in this group. Three possible sources for a reduced O_2 delivery to muscle cells would be: a ventilation-perfusion mismatch ($\dot{V}_A:\dot{Q}$), a veno-arterial shunt, or a diffusion limitation from the alveolus to pulmonary capillary (12). In turn, each of these factors could be limited by the amount

of air that is exposed to the alveoli for gas exchange. Three possible sources for a ventilatory limit are: the mechanical resistance of the airways and chest wall compliance, the energetics of moving air into and out of the lungs, and respiratory muscle (RM) fatigue.

Ventilation may be limited by mechanical factors. Dempsey (11) recorded transpulmonary pressures and flow volume-loops ($\dot{V}$-V) during maximum exercise and during a test of MVV. There were no differences in the $\dot{V}$-V loops. However, the transdiaphragmatic pressures (Pdi) recorded at the same time were 4.5 times greater during the MVV, demonstrating that increased force did not increase flow. Airway resistance also presents another mechanical limitation. Bye et al (7) reported that athletes breathing less dense gas, (80% Helium: 20% O_2) were able to achieve higher ventilation than while breathing air and, in addition, previously recorded hypoxemia of these athletes was corrected. Hussain et al (17) concurred with this observation of less work of breathing, by reporting a decrease of 40% in Pdi (gastric pressure minus pleural pressure) measured during moderate exercise while breathing HeO_2. In addition, Tenney and Reese (42) reported higher ventilation for specific respiratory endurance tests while breathing HeO_2 in comparison to air. Therefore, mechanical factors may limit the maximal ventilation that can be reached which may keep the O_2 supply below the O_2 demand of highly trained athletes.

A second factor that possibly limits ventilation during athletic performance is the use of energy by the RM. By analyzing expired gases during different levels of $\dot{V}_E$, the

amount of O_2 consumed by the respiratory muscles ($\dot{V}O_2$resp) can be estimated (4). Bye et al (6) reported the range of estimated $\dot{V}O_2$resp to be from 2 to 9 ml of O_2 per litre of $\dot{V}_E$, which would be from 320 ml to 1440 ml of O_2 at a $\dot{V}_E$ of 160 l/min. Similar results are obtained when $\dot{V}O_2$resp is estimated by measuring blood flow to the RM. Extrapolating from the research on dogs by Robertson et al (35), Bye et al estimated that blood flow to the RM can reach approximately 8 l/minute. Assuming an O_2 content of venous blood from the respiratory muscles of 15 Vols%, the $\dot{V}O_2$resp would be 1.2 l/min or approximately 25% of the maximal oxygen consumption (VO_2max). Otis (32), Shephard (41) and Margaria (22) have each agreed that there is a level of V_E, the critical ventilation, above which the increased amount of O_2 associated with increased $\dot{V}_E$, is used solely by the RM. The values offered by these authors for this critical ventilation are 140 l/min, 135 l/min and 120-170 l/min respectively, depending on the individual subject.

A third factor possibly limiting ventilation is fatigue of the respiratory muscles. Several studies (1, 15, 28) have reported fatigue of the diaphragm, intercostal, and sternomastoid muscles. Direct measurements of these muscles with electromyographic (EMG) recordings show that after repeated electrical stimulation and/or endurance breathing tests, there is a decrease in the high/low frequency power ratio of the diaphragm as well as decreased contraction force of the sternomastoid. Other studies (3, 21, 25) have used indirect measurements of fatigue such as: decreased lung capacities, decreased maximal respiratory muscle strength, and decreased respiratory

endurance performance to demonstrate fatigue after exhaustive ventilatory or aerobic performances. Thus, fatigue of the respiratory muscles would result in a decreased ability of these muscles to maintain a specified level of ventilation.

As Bye et al (6) point out, a consequence of inadequate ventilation is hypoxemia which causes reduced maximal working capacity, reduced maximal oxygen consumption ($\dot{V}O_2max$) and decreased endurance time. An approach to increasing the amount of O_2 available to working muscles during exercise, would be to either increase the level of ventilation and/or decrease the amount of O_2 used by the RM. Increasing the efficiency of the working muscles reduces the amount of oxygen required for a given task and delays the onset of fatigue. The respiratory muscles constitute only 6% of total body weight but they may theoretically consume up to 25% of the $\dot{V}O_2max$ during exercise (6). Improvement in the efficiency of these muscles may be reflected as improved endurance performance.

Saltin and Gollnick (38) point out that the fibre type of the diaphragm and the intercostal muscles is very similar to the fibre type of the vastus lateralis which contains approximately 50% of both slow twitch and fast twitch fibers. Sharp and Hyatt (40) have reviewed the similarity of the mechanical and electrical properties of respiratory muscles with other skeletal muscles. Therefore, the same adaptation to endurance training that Secher et al (39) describe for skeletal muscle, should occur in the respiratory muscles. Decreasing the amount of O_2 required by the RM for a given

ventilation , should make more O_2 available to working limb muscles and this could result in increased aerobic performance.

In 1976, Leith and Bradley (20) demonstrated that the RM could be trained for strength and endurance. One group of subjects (age = 31 years), followed a five week endurance training protocol consisting of normocarbic hyperpnea and increased their sustained ventilatory capacity (SVC) by 19%. The strength training group showed a 55% increase in maximum inspiratory (MIP) and expiratory pressures (MEP). Keens et al (18) also demonstrated an improvement in RM endurance when four subjects (age = 29 years) increased their maximal sustained ventilatory capacity (MSVC) by 22%. Recently, Belman & Gaesser (2), have reported an increase of 21% in RM endurance in elderly subjects (age = 70 yr) following eight weeks of isocapnic hyperpnea training. In 1987, Morgan et al (27) provided the first report of RM training of moderately fit athletes (VO_2max = 50 ml/kg/min, age = 24 yr). Prior to and following four weeks of normocapnic hyperpnea training, they measured $\dot{V}O_2max$, cycling endurance, MVV_{15} and an endurance breathing test (100% of MVV for time). After training there was an increase in the MVV as well as an increase in the duration of the 100% MVV, but no significant change in the VO_2max or cycling endurance test. Unfortunately, the training intensity and duration are unclear. Their protocol states that the subjects would begin training at an intensity of 181 l/min (85% of MVV), for the maximum sustainable duration, and yet, the reported average ventilation was 165 l/min for the first week.

In addition, the MVV_{15} is a measure of the sprint performance of the RM rather than an endurance measure.

The purpose of this study was to determine the effects of four weeks of standardized isocapnic hyperpnea training on both the endurance performance of the respiratory muscles as well as overall aerobic performance. The first issue to be addressed was whether the respiratory muscle endurance could be improved in highly trained cyclists ($\dot{V}O_2max$ > 60 ml/kg/min). Secondly, do four, 30 minute sessions of hyperpnea training per week increase the $\dot{V}O_2max$ or the endurance cycling performance of highly trained cyclists?

METHODS AND PROCEDURES

Subjects

Ten well-trained male cyclists (mean $\pm$ SD; age = 22.0 $\pm$ 3.4 yr, height = 176.0 $\pm$ 6.4 cm, weight 71.3 $\pm$ 6.8 kg, $\dot{V}O_2max$ = 66.1 $\pm$ 4.6 ml/kg/min) volunteered for this study. The three criteria for inclusion in the study were: a VO_2max greater than 60 ml/kg/min, normal values for spirometry, and an active participation in cycling events. Informed consent was obtained from each subject. Two groups of five subjects were assigned to control for experimental conditions. The training group participated in isocapnic hyperpnea exercise and the control subjects participated in all testing sessions but not in the RM training program. The subjects continued with their regular aerobic training programs and were required to submit a record of the number of kilometers they cycled each week of the project.

Maximum Sustained Ventilatory Capacity

The maximum sustained ventilatory capacity (MSVC) test was used to measure respiratory muscle endurance. The MSVC is determined by measuring the maximum ventilation a person can sustain for a specified time. The MSVC test duration for this study was 10 minutes as described by Belman & Gasser (2). Prior to the test, the subjects breathed on the ventilatory endurance apparatus for two minutes at 50% of their maximal exercise ventilation ($\dot{V}_Emax$) followed by one minute of rest. During the

first two minutes of the test the air flow was gradually increased to the maximum tolerable by the subject. For the next 8 minutes, the air flow was adjusted to maintain the maximum possible $\dot{V}_E$ the subject could tolerate. To ensure the baseline MSVC values were maximal, the test was performed by each subject until two tests, separated by 48 hours, were within 5% of each other. The mean $\dot{V}_E$ obtained during the last eight minutes of the test was the baseline measurement. To determine the number of tests required for a reliable baseline measurement, 6 additional normal subjects completed three MSVC tests on separate days within a two week period.

Ventilatory Endurance Apparatus

The ventilatory endurance apparatus (Figure 1, page 10) was designed to allow isocapnic hyperpnea for testing and training the endurance of the RM. On the inspired side of the circuit, a vacuum pump (Bodine Electric, Chicago, IL) supplied a variable air flow which passed through an air flow meter (Vacumetrics, Ventura, CA) and then into a 13.5 litre mixing chamber. 100% CO_2 was added at the rate of 3.4 to 4.25 l/minute to maintain the fractional concentration of mixed expired CO_2 ($FECO_2$) at each subjects' resting level (approximately 5%). A 9-liter Respirometer (Collins, Boston, MA) provided the visual reference for the target ventilation for the subject. The subject was instructed to keep a mark on the bell of the respirometer below the water reservoir level. A 5-litre anaesthesia bag was included on the inspired side of the system to provide a dampening effect by expanding while a subject was swallowing.

Distal to this bag was a sampling tube connected to a CO_2 analyzer (Medical Graphics, St. Paul, MN) to measure the fractional concentration of inspired CO_2 (F_ICO_2). The subject breathed through a low resistance 2-way valve (Hans Rudolph #2700 K.C., MO.) and the expired gas passed through a heated pneumotachograph (Model #5, Fleisch, Switzerland) to measure $\dot{V}_E$. A temperature gauge recorded expired gas temperature. $\dot{V}_E$ was processed on line by an IBM microcomputer (Armonk,N.Y.). The gas analyzers were calibrated with air and calibration gas before each test. The pneumotachograph was calibrated at the maximum volume of 230 l/min as measured by air flow meter. The resistance of the circuit was measured over the range of air flow from 40 to 200 l/min (see Appendix E, page 58).

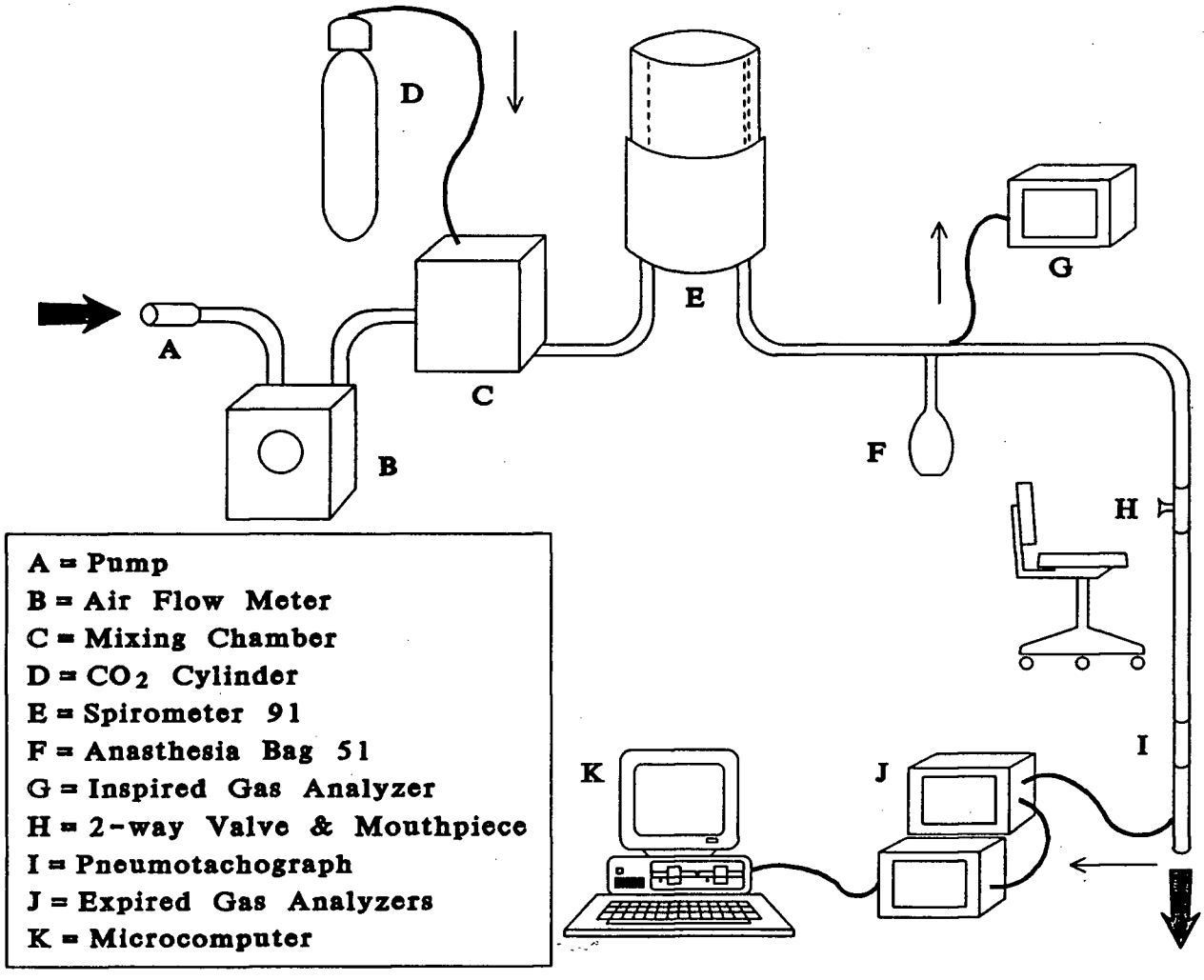

Figure 1: Ventilatory Endurance Apparatus for testing and training respiratory muscles. Arrows indicate direction of air flow.

Pulmonary Function Tests

Pulmonary function tests were performed before and after the 16 training sessions. Forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), and MVV_{12} were measured and analyzed using the Medical Graphics computerized spirometer system (St. Paul, MN.) with the associated 1070 software package. The highest of three values for the MVV_{12} (within 10% of each other) was recorded as a measure of the sprint performance of the ventilatory system.

Maximal Aerobic Capacity ($\dot{V}O_2max$)

Measuring the $\dot{V}O_2max$ served two purposes for this study: the initial test was to ensure the subjects had a $\dot{V}O_2max$ greater than 60 ml/kg/min, and the test following training was an index of whether the aerobic fitness of the subjects had changed during the project. The incremental cycle ergometer test was performed on an electronically braked cycle ergometer (Mijnhardt, Holland) using a ramp protocol with the work beginning at 0 watts and the power increasing by 30 watts per minute. Expired air was measured and analyzed by either the Medical Graphics system with the 2001 software package or with O_2 and CO_2 analyzers (Beckman OM11, LB2,Fullerton, CA), a heated pneumotachograph (Fleisch, Switzerland) to measure $\dot{V}_E$ and data was processed on-line with an IBM microcomputer. Heart rate (HR) was recorded using direct lead ECG (Lifepack 6, Physio Control Canada, Agincourt, Ontario). Criteria

for attaining $\dot{V}O_2$max was a plateau in $\dot{V}O_2$ with an increased workload, a respiratory exchange ratio (RER) greater than 1.15, and HR greater than 180 beats per minute (90% predicted maximum heart rate). Calibration of the pneumotachograph was performed with a 3-liter syringe and the gas analyzers were calibrated with air and calibration gases prior to each test.

Performance Cycle Test

Each subject performed a cycle ride to exhaustion at a power which represented 90% of the maximum work rate previously obtained during the $\dot{V}O_2$max test. The purpose of the performance test was to simulate a cycle ride while standardizing the external environment. Previous experience in this laboratory with similar subjects has shown that the approximate time for a test at this work level is from 6 to 10 minutes, a duration similar to the MSVC test and MSVC training times. Following a 3 minute warm up, resistance was increased over 10 seconds until the predetermined work rate was attained. From this point, expired ventilation ($\dot{V}_E$tlim) was recorded as well as the time to exhaustion (tlim). The criterion for the end of the test was the inability to maintain the minimum pedal frequency of 60 RPM for 3 consecutive revolutions. Subjects were not aware of the elapsed time.

Respiratory Muscle Endurance Training Protocol

To improve the endurance performance of the RM a volume overload technique, isocapnic hyperpnea, was selected. The subjects attended three or four training sessions per week for a total of 16 sessions. Each session consisted of three, eight minute work intervals of isocapnic hyperpnea alternated with eight minute intervals of rest. The training overload was a combination of increasing both ventilation and duration of the work intervals. Initially, the target ventilation for each work interval was the ventilation each subject achieved during the initial MSVC test. Progressively, the subjects were able to maintain this target ventilation for each of the three work intervals. To provide a training stimulus, the target ventilation was then increased to a level that could only be maintained for the first work interval of a training session and the subject's new goal was to maintain this larger ventilation for all three work intervals. Following the eighth training session, the duration of both the work and rest intervals was increased to 10 minutes each.

STATISTICAL ANALYSES

A repeated measures analysis of variance was used to test the reliability of the three MSVC tests performed by 6 subjects. Analysis of variance was used to test the similarity of the experimental and control groups prior to RM endurance training for the following variables: age, height, weight, FVC, FEV_1, MVV_{12}, MSVC, $\dot{V}O_2max$, $\dot{V}_Emax$, tlim, and $\dot{V}_Etlim$.

To determine the effects of the RM training, mean group pre and post-training differences were determined by multiple analysis of variance for the variables in the following groups:

1. Pulmonary functions: FVC, FEV_1, MVV
2. Maximal aerobic capacity test: $\dot{V}O_2max$, $\dot{V}_Emax$
3. Endurance cycle test: tlim, $\dot{V}_Etlim$.

Analysis of variance was used to determine the significance of the difference in the MSVC test between the groups. The data analysis was performed with the statistical package, SYSTAT, version 4 (43). The level of significance for each test was $P < 0.05$. Data are expressed as mean $\pm$ SD.

RESULTS

There were no differences between the training and control groups for age, height, weight, or $\dot{V}O_2max$ (Appendix B, page 49). All 10 cyclists completed the study and maintained the same average number of kilometers cycled per week (221 $\pm$ 181 km). There were no differences between groups for FVC, FEV_1, or MVV either pre or post-training (Table 1, page 16). In addition, both the experimental and control subjects had similar pre-training values for the MSVC test (Table 1 and Figure 2, page 18). For both the incremental and endurance cycle tests, the $\dot{V}O_2max$, $\dot{V}_Emax$, $\dot{V}_Etlim$ and tlim were similar for each group both before and after training (Table 2, page 17). There was no change in the maximal work rates achieved by either the control or the experimental subjects (387 $\pm$ 47 vs 390 $\pm$ 34; 395 $\pm$ 30 vs 393 $\pm$ 22 watts). Following 16 training sessions, the experimental group demonstrated a significant increase in MSVC (155.4 $\pm$ 11.2 vs 173.9 $\pm$ 11.6 l/min, $p = 0.004$). The control groups values were not different (155.1 $\pm$ 26 vs. 149.5 $\pm$ 34 liters) following the four week training period (Figure 3).

The tests of reproducibility indicated there were no significant differences between the means of the ventilation for the three MSVC tests performed by the six additional subjects (test 1= 141.3 $\pm$ 15.9; test 2= 150.3 $\pm$ 22.4; test 3= 153.8 $\pm$ 34.0 l/min, $p = 0.132$).

TABLE 1

PRE AND POST-TRAINING VENTILATION VARIABLES

(MEAN ± SD * p = 0.004)

		CONTROL		EXPERIMENTAL	
VARIABLE		PRE-TRAIN	POST-TRAIN	PRE-TRAIN	POST-TRAIN
FVC		5.7	5.6	5.5	5.7
(liters)	±	0.7	0.7	0.9	1.0
FEV_1		4.8	4.7	4.4	4.5
(liters)	±	0.6	0.7	0.3	0.4
MVV_{12}		215.2	223.3	205.5	216.3
(l/min)	±	20.0	25.7	15.2	18.8
MSVC		155.1	149.5	155.4	173.9*
(liters)	±	25.8	33.9	11.2	11.6

FVC= Forced vital capacity

FEV_1= Forced expiratory volume in one second

MVV_{12}= Maximal voluntary ventilation measured for 12 seconds

MSVC= Maximum sustained ventilatory capacity

TABLE 2

PRE AND POST-TRAINING EXERCISE VARIABLES

(MEAN ± SD)

	CONTROL		EXPERIMENTAL	
VARIABLE	PRE-TRAIN	POST-TRAIN	PRE-TRAIN	POST-TRAIN
$\dot{V}O_2max$ (ml/kg/min)	68.0 ± 6.6	67.1 5.8	64.2 1.9	65.8 4.8
$\dot{V}_E max$ (l/min)	171.4 ± 35.5	167.5 21.4	177.0 21.6	177.1 12.6
$\dot{V}_E tlim$ (l/min)	174.0 ± 29.2	176.3 26.8	176.0 16.2	178.5 19.3
tlim (seconds)	328.6 ± 99.0	342.4 79.6	342.2 74.9	427.8 226.1

$\dot{V}O_2max$= Maximal oxygen consumption

$\dot{V}_E max$= Maximal ventilation during VO_2max test

$\dot{V}_E tlim$= Maximal ventilation during performance cycle test

tlim= Time to exhaustion (cycle endurance test)

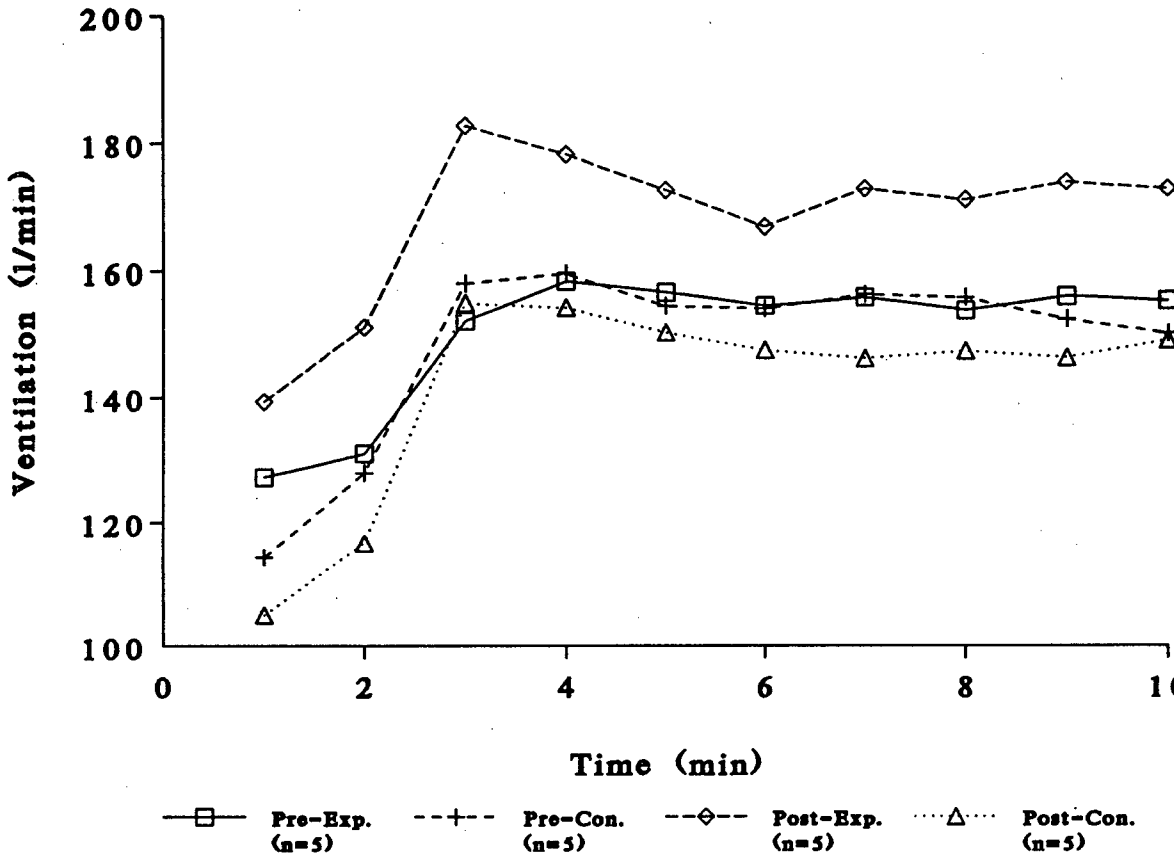

Figure 2: Mean ventilation for each minute of the MSVC test. The mean of the last 8 minutes was greater ($p = 0.004$) for the experimental subjects following training.

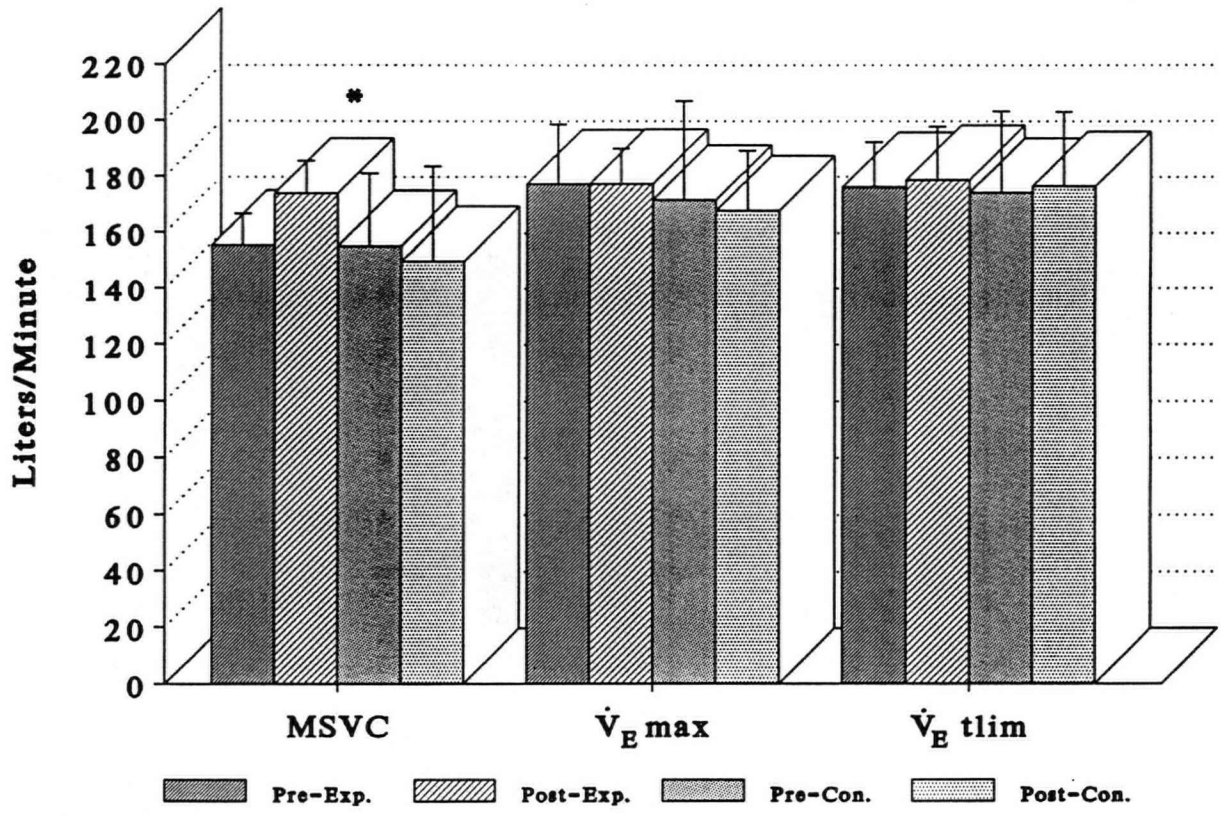

Figure 3: Test ventilation for experimental and control subjects pre and post-training. Note: * = increase in MSVC for experimental subjects ($p = 0.004$)

DISCUSSION

This study shows that 16 training sessions of isocapnic hyperpnea increased the respiratory muscle endurance of highly trained cyclists. The 5 experimental subjects were able to increase their maximal sustained ventilatory capacity by 12%, demonstrating that their respiratory muscle endurance was not maximal as a result of their regular cardiovascular training programs. Since the control subjects did not increase their RM endurance, we can assume the increase for the experimental subjects was due to the RM training. Athletes involved in sports requiring a high degree of aerobic fitness have greater respiratory muscle endurance than non-athletes as a result of the high levels of ventilation they maintain during their cardiovascular workouts (25). The increased respiratory muscle endurance of the athletes is thought to be as a result of training rather than genetic predisposition (23). Also, a prospective study by Robinson and Kjeldgaard (36) demonstrated a 16% improvement in ventilatory muscle endurance in 11 sedentary subjects following 20 weeks of supervised running.

The 12% increase in MSVC in this study is not as large an increase as demonstrated by Leith and Bradley (20), Belman and Gaesser (2),or Keens et al (18). The studies reported increases of 21%, 20% and 22% respectively. Leith and Bradley (20) reported an increase of 21% but the endurance breathing test (SVC) they used was a composite of 8 to 10 points of different levels of ventilation against time and therefore not comparable to the MSVC used in this study. The mean age of the

subjects of the Belman and Gaesser study was 67 years and the ventilation during the MSVC were only 62-64% of the MVV_{12} while the pre-training value for the subjects in this study was 75% of their MVV_{12}. The larger amount of improvement reported in the study by Keens et al, was possibly because their subjects (age = 28.3 $\pm$ 2.6 yr) had 30 training sessions compared to 16 for the subjects in this study.

The MSVC expressed as a percent of $V_E$max increased from 88% pre-training to 98% post-training (Figure 4, page 22). The tidal volume during the pre-training MSVC for the E subjects was 2.56 $\pm$ 0.22 l and the respiratory rate (RR) was 62 $\pm$ 3 breaths/min and the post-training values increased to a V_T of 3.38 $\pm$ 0.2 l and decreased to an RR of 53 $\pm$ 3 breaths/min. The post-training breathing patterns are very similar to the V_T and RR of $\dot{V}_E$max (3.4 $\pm$ 0.6 l and 54 $\pm$ 7 breaths/min respectively).

As anticipated, the MVV_{12} did not change following the hyperpnea training. Unlike the studies of Leith and Bradley (20) and Morgan et al (27), the subjects in this study were not encouraged to train at the MVV_{12} ventilation. The breathing patterns selected by the subjects in this study illustrate the dissimilarity of the MSVC and the MVV_{12} tests. In this study, the ventilation for the MSVC and the MVV_{12} were 174 $\pm$ 12 and 216 $\pm$ 19 l/min, the V_T were 3.4 $\pm$ 0.2 and 2.2 $\pm$ 0.2 l and the respiratory rates were 54 $\pm$ 3 and 100 $\pm$ 10 breaths/min respectively. Klas and Dempsey (29) measured the end expiratory lung volume (EELV) during an MVV test and during maximal exercise and found the MVV test was performed at a higher lung volume. We did not

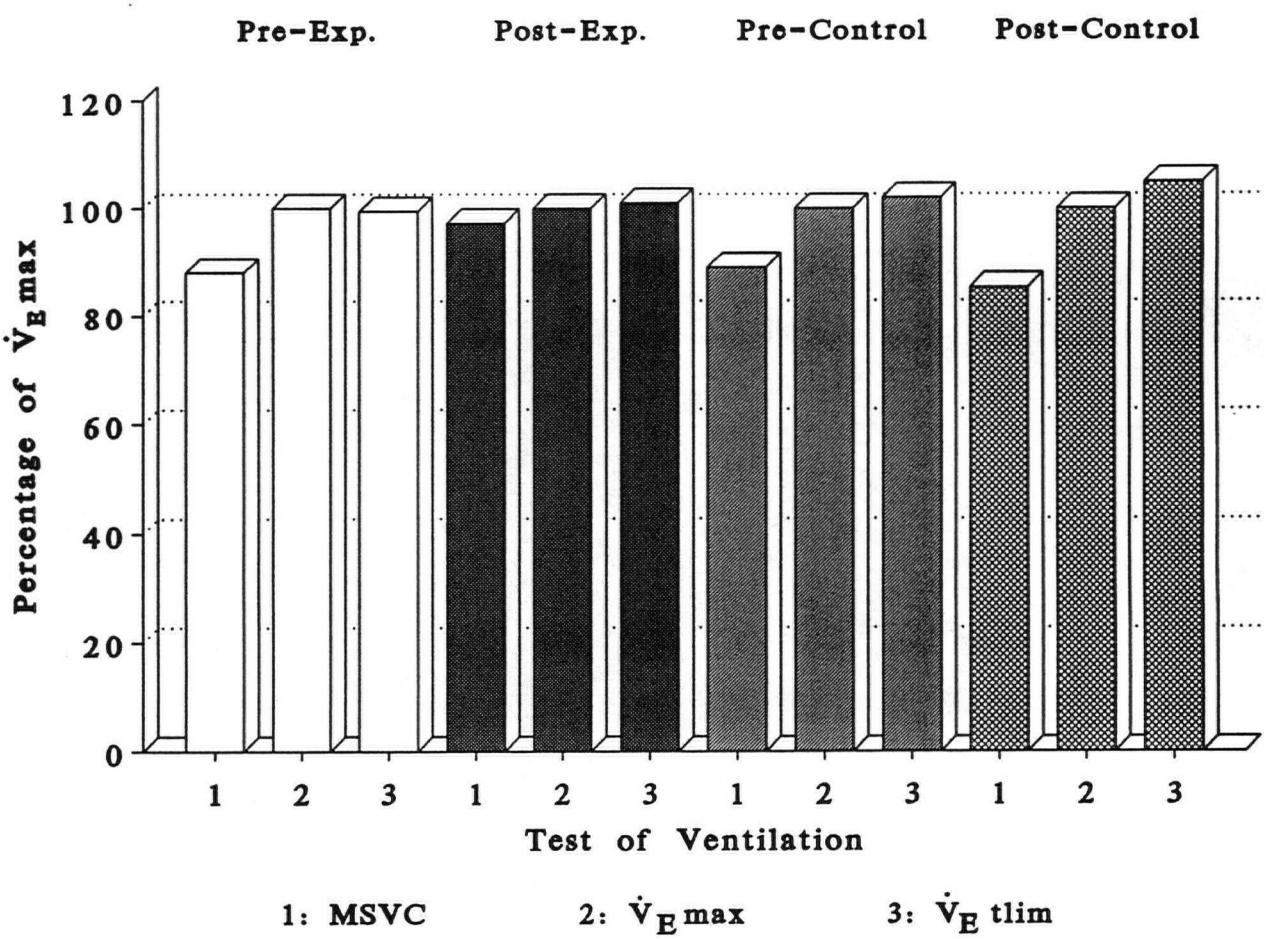

Figure 4: Test ventilation expressed as a percentage of $\dot{V}_E$max for control and experimental subjects pre and post-training. The x-axis indicates the test: #1= MSVC; #2= Ventilation during $\dot{V}O_2$max ($\dot{V}_E$max); #3= Ventilation during cycle endurance ($\dot{V}_E$tlim).
Note: MSVC increased from 88% to 98% for the experimental subjects after training.

measure the EELV, but since the breathing patterns were dissimilar between the MVV and the MSVC, we can assume the EELV was different. However, since the breathing patterns for the MSVC and $\dot{V}_E$max were similar, the respiratory muscle training may have been performed at a lung volume comparable to the exercise ventilation. If the hyperpnea training program had been more specific and begun at the exercise ventilation rather than at 88% of $V_E$max, perhaps there may have been an increase in the performance cycle test due to the energy sparing effect of the RM at the exercise ventilation.

The $\dot{V}O_2$max of our subjects did not change following hyperpnea training and these results are consistent with the studies of Morgan et al (27) and Belman and Gaesser (2). To increase the $\dot{V}O_2$max, it would be necessary to increase the amount of O_2 available to working muscles and this can be accomplished by increasing either the cardiac output or the systemic arteriovenous oxygen difference. The type of exercise necessary to effect these changes would be activities which require rhythmic contractions of large muscle groups. Following exercise of sufficient duration and intensity, the $\dot{V}O_2$max will most likely improve as a result of increased cardiac output (9). Since RM training involves a small group of muscles, there is no reason to anticipate an adaptation in the central circulatory response to exercise. Adaptation to RM training is most likely a result of local changes in the oxidative metabolic capacity of the trained muscles and a decrease in local peripheral resistance.

There were no significant changes in the performance cycling test measurements and again these results are similar to the study of Morgan et al (27). Tlim increased in this study by 86 seconds (from 342 $\pm$ 75 to 428 $\pm$ 226 seconds), but these results were affected by one subject who increased from 459 to 801 seconds. Since the $\dot{V}O_2$ and $\dot{V}_E$tlim were very similar to the $\dot{V}O_2$max and $\dot{V}_E$max during the $\dot{V}O_2$max test, perhaps other variables such as motivation, buffering capacity and fiber type may be responsible for the variability in the tlim. However, it would be reasonable to assume that increasing the efficiency of the RM would result in a decrease in the amount of total O_2 they would need for the same level of work at a submaximal intensity (9). Theoretically, this training effect would increase the amount of O_2 available to other working muscles and delay fatigue during maximal exercise. A more suitable test of assessing the response to RM training might be to measure heart rate, $\dot{V}_E$, and $\dot{V}O_2$ at 60% of $\dot{V}O_2$max.

Neither $\dot{V}O_2$resp nor RM fatigue were measured during this study. Under ideal circumstances, measuring these two variables during a hyperpnea training protocol would provide information on the intensity and duration of training necessary to effect metabolic adaptation of the RM.

In conclusion, four weeks of isocapnic hyperpnea exercise increased the respiratory muscle endurance of five highly trained male cyclists, but did not effect a change in maximal exercise performance.

REFERENCES

1 Bai TR, Rabinovitch BJ, and Pardy RL: Near-maximal voluntary hyperpnea and ventilatory muscle function. J. Appl. Physiol. 57(6):1742-1748, 1984.

2 Belman MJ, and Gaesser GA: Ventilatory muscle training in the elderly. J. Appl. Physiol. 64(3):889-905, 1988.

3 Bender PR, and Martin BJ: Maximal ventilation after exhausting exercise. Med. Sci. Sports Exerc. 17(1):164-167, 1985.

4 Bradley ME, and Leith DE: Ventilatory muscle training and the oxygen cost of sustained hyperpnea. J. Appl. Physiol. 45(6):885-892, 1978.

5 Brooks GA, Fahey RD: Exercise Physiology: Human Bioenergetics and its Applications. Wiley & Sons, 1984. Chapter 13:271-278.

6 Bye PT, Esau SA, Walley KR, Macklem PT, Pardy RL.: Ventilatory muscles during exercise in air and oxygen in normal men. J. Appl. Physiol.: REEP. 56(2): 464-471, 1984.

7 Bye PTP, Farkas FA, Roussos Ch: Respiratory factors limiting exercise. Ann. Rev. Physiol. 45:439-451, 1983.

8 Celli RC.: Clinical and physiologic evaluation of respiratory muscle function. Clin Chest Medicine. 10 (2):199-214, 1989.

9 Clausen JP.: Effect of physical training on cardiovascular adjustments to exercise in man. Physiol. Review. 57:770-815, 1977.

10 Collett PW, Engel LA.: Influence of lung volume on oxygen cost of resistive breathing. J. Appl. Physiol. 61(1):16-24, 1986.

11 Dempsey JA: Is the lung built for exercise? Med. Sci. Sports Exerc. 18(2):143-155, 1986.

12 Dempsey JA, Hanson PG, and Henderson KS: Exercise-induced arterial hypoxemia in healthy human subjects at sea level. J. Physiol. 355:161-175, 1984.

13 Dodd DS, Collett PW, Engel LA.: Influence of inspiratory flow rate and frequency on O_2 cost of resistive breathing in humans. J. Appl. Physiol. 65(2):760-766, 1988.

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

15 Grassino A, Gross D, Macklem PT, Roussos Ch, Zagelbaum G: Inspiratory muscle fatigue as a factor limiting exercise. Clin. Resp. Physiol. (formerly, Bull. Europ. Physiopath. Resp.) 15:105-111, 1979.

16 Hopkins SR, McKenzie DC: The hypoxic ventilatory response and arterial desaturation during heavy work. J. Appl. Physiol. (in press 1989).

17 Hussain SNA, Pardy RL, Dempsey JA: Mechanical impedance as determinant of inspiratory neural drive during exercise in humans. J. Appl. Physiol. 59(2):365-375, 1985.

18 Keens TG, Krastins IRB, Wannamaker EM, Levison H, Crozier DN, and Bryan AC: Ventilatory muscle endurance training in normal subjects and patients with cystic fibrosis. Am. Rev. Resp. Disease. 116:853-860, 1977.

19 Klas JV, Dempsey JA.: Voluntary versus reflex regulation of maximal exercise flow:volume loops. Am. Rev. Resp. Dis. 139:150-156, 1989.

20 Leith DE, Bradley M: Ventilatory muscle strength and endurance training. J. Appl. Physiol. 41(4):508-516, 1976.

21 Loke J, Mahler DA, and Virgulto JA: Respiratory muscle fatigue after marathon running. J. Appl. Physiol. 52(4):821-824, 1982

22 Margaria R, Milic-Emili G, Petit JM, Cavagna G: Mechanical work of breathing during muscular exercise. J. Appl. Physiol. 15:354-58, 1960.

23 Martin BJ, Chen HI: Ventilatory endurance in athletes: a family study. Int. J. Sports Med. 3:100-104, 1982.

24 Martin B, Heintzelman M, Chen H.: Exercise performance after ventilatory work. J. Appl. Physiol.: REEP 52(6): 1581-1585, 1982.

25 Martin BJ, Stager JM: Ventilatory endurance in athletes and non-athletes. Med. Sci. Sports Exerc. 13(1):21-26, 1981.

26 McCool FD, Tzelepis GE, Leith DE, Hoppin JR. FG.: Oxygen cost of breathing during fatiguing inspiratory resistive loads. J. Appl. Physiol. 66(5): 2045-2055, 1989.

27 Morgan DW, Kohrt WM, Bates BJ, and Skinner JS: Effects of respiratory muscle endurance training on ventilatory and endurance performance of moderately trained cyclists. Int. J. Sports Med. 8(2):88-93, 1987.

28 Moxham J, Wiles CM, Newham D, and Edwards RHT: Sternomastoid muscle function and fatigue in man. Clin. Sci. 59:463-468, 1980.

29 Ogilvie CM, Stone RW, Marshall R.: The mechanics of breathing during the maximum breathing capacity test. Clin Sci 14:101-107, 1955.

30 Olafsson S, Hyatt RE: Ventilatory mechanics and expiratory flow limitation during exercise in normal subjects. J. Clin. Invest. 48:564-573, 1969.

31 Otis AB: The work of breathing. Physiol. Rev. 34:449-58, 1954.

32 Otis AB: The work of breathing. Handbook of Physiology: Respiration I. Section 3. Vol. 1 17:463-476, 1964.

33 Pardy RL, Leith B.: Ventilatory muscle training. Resp. Care 29:278-284 1984.

34 Powers SK, and Williams J: Exercise-induced hypoxemia in highly trained athletes. Sports Medicine. 4:46-53, 1987.

35 Robertson Jr.CH, Foster GH, Johnson Jr. RL: The relationship of respiratory failure to the oxygen consumption of, lactate production by, and distribution of blood flow among respiratory muscles during increasing respiratory resistance. J. Clin. Invest. 59:31-42, 1977.

36 Robinson EP, Kjeldgaard JM: Improvement in ventilatory muscle function with running. J. Appl. Physiol:REEP 52(6):1400-1406, 1982.

37 Roussos C, Fixley M, Gross D, and Macklem PT.:Fatigue of inspiratory muscles and their synergic behavior. J. Appl. Physiol:REEP 46(5): 897-904, 1979.

38 Saltin B, Gollnick PD.: Skeletal muscle adaptability: significance for metabolism and performance. Handbook of Physiology: Section 10, Skeletal muscle. Chap 19:555-631, 1983.

39 Secher NH, Mizuno M, Saltin B.: Adaptation of skeletal muscles to training. Bull. Eur. Physiopathol. Respir. 20:453-457, 1984.

40 Sharp JT., Hyatt RE.: Mechanical and electrical properties of respiratory muscles. Handbook of Physiology: The respiratory system III, Mechanics of breathing, Part 2. Chap 23:389-409, 1986.

41 Shephard RJ: The maximum sustained voluntary ventilation in exercise. Clin. Sci. 32:167-176, 1967.

42 Tenney SM, Reese RE: The ability to sustain great breathing efforts. Resp. Physiol. 5:187-201, 1968.

43 Wilkinson, L. SYSTAT: The System for Statistics, Evanston, IL: SYSTAT, Inc. 1988.

APPENDIX A: Review of Literature

REVIEW OF LITERATURE

The following section will be a review of the research supporting the view that the respiratory system may contribute to limitation of performance of elite athletes at maximal exercise levels. Three possible sources for a ventilatory limit to exercise are: the mechanical resistances of the airways and chest wall, the energetics of respiratory muscles at high levels of ventilation, and respiratory muscle fatigue.

Mechanics

Olafsson and Hyatt (30) published the results of a study in 1969 with the following conclusion: "The mechanical properties of the lung do not appear to limit ventilation during exhausting exercise in normal subjects." This conclusion was based on measurements of esophageal pressure and lung volume on subjects performing a maximum breathing capacity (MBC) test at rest, and during short term running to exhaustion on a treadmill. The MBC requires the subject to breathe as much air as possible in 15 or 20 seconds while CO_2 is added to the inspired air. The subjects were 10 healthy, sedentary males (age 36 $\pm$ 8.5 years). During the treadmill exercise the average ventilation was 120 $\pm$ 13.7 l/min and the mean inspiratory and expiratory transpulmonary pressures were -30 and 6 cmH_2O respectively. Both the measurements of maximal inspiratory flow (5.6 vs 9.5 l/sec) and maximal expiratory flow (9.5 vs. 11.3

l/sec) were less during exercise than the values obtained during the MBC test. Olafsson and Hyatt concluded that ventilation during near-maximal exercise remained efficient.

There are two reasons the conclusion of Olafsson and Hyatt (30) should be re-assessed: First, the sedentary activity level of their subjects may limit the extent to which their findings may be generalized and second, the MBC test used for comparison with exercise ventilation may be inappropriate due to the lack of specificity between the test and exercise ventilation.

For example, the subjects who participated in the research of Dempsey et al (12) and Hopkins and McKenzie (16) were 16 and 12 highly trained athletes whose $\dot{V}O_2max$ was 72 ml/kg/min and 63 ml/kg/min and whose maximal ventilation during exercise was 157 l/min and 158 $\pm$ 19.6 l/min respectively. It does not seem reasonable to generalize from the data of sedentary subjects to these extremely fit individuals. In fact, 24 of these 28 subjects became hypoxic during maximal exercise. The authors did not find inadequate ventilatory drives or hypoventilation as a cause of this hypoxemia.

In addition to the sedentary nature of Olafsson's subjects, Klas and Dempsey (19) have demonstrated that the MVV test is not appropriate for making comparisons with exercise ventilation. Olafsson and Hyatt used the MBC test which has a 15 second duration similar to the MVV test. Klas and Dempsey (19) calculated lung volumes by measuring end expiratory lung volume (EELV) in five subjects and found that during exercise the EELV was 2.23 $\pm$ 0.2 liters while during the MVV test it was 2.74 $\pm$ 0.3 liters. Therefore, this data demonstrates that the breathing response of exercise is

performed at a lower lung volume than during a voluntary breathing test such as the MVV. Thoracic pressures, flow:volume measurements and minute ventilation recorded during voluntary hyperventilation (MVV test) should not be compared to measurements recorded during the involuntary hyperpnea of exercise. Breathing at a higher lung volume allows maximal expiratory pressures to be higher than during exercise and also the pressure:volume relationships are different at different lung volumes. Also, since the MVV test duration is only 12-15 seconds, it is not the correct test to use when investigating the possibility that ventilation is a limiting factor of peak athletic performance since aerobic exercise continues longer than a few seconds. Also, ventilation during exercise is generally lower (80% of MVV) than during the MVV test mainly due to a lower breathing frequency and larger tidal volume. The very small amount of end tidal CO_2 exhaled during an MVV is an indication the rapid shallow breathing pattern does not allow adequate ventilation and therefore is inappropriate for aerobic activity. Klas and Dempsey (19) suggest the efficient replication of exercise ventilation can be accomplished by visual feedback allowing the control of lung volume, pressure:volume, and breath-timing characteristics.

The ventilatory reserve discussed by Brooks and Fahey (5) is the difference between the ventilation measured during the MVV test and maximal exercise ventilation. Since the MVV is a test of the sprint performance of the RM and not an appropriate test for sustained ventilatory capacity, perhaps the importance of a ventilatory reserve should be re-evaluated. To evaluate the exercise ventilation of

athletes, a test protocol should be used that requires the respiratory system to closely mimic its function during actual performance. Incremental ergometer tests are used to evaluate cardiovascular and metabolic performance and perhaps the maximum ventilation is actually reached with the same protocol.

ENERGETICS

A decrease in VO_2 for a given workload is the best indicator of increased muscular efficiency reflecting a training response. Unfortunately, measuring the $\dot{V}O_2$ of the respiratory muscles ($\dot{V}O_2$resp) in humans can only be estimated by indirect measurements. Most researchers (13,10,41,32) measure the $\dot{V}O_2$ for the entire body at different ventilation and then assume the difference in $\dot{V}O_2$ between these different ventilation is due to the $\dot{V}O_2$resp. The reported (7) range of $\dot{V}O_2$resp for ventilation over 100 l/min is from 2 to 8 ml O_2/liter and Otis (32) found large inter-subject variability when four different investigators measured the $\dot{V}O_2$resp at 70 l/min and reported a range of 50 to 375 ml/min. As an example of intra-subject variability, during a study by Otis (31), one subject breathed at the rate of 215 and 224 l/min during two MVV tests and the reported $\dot{V}O_2$resp for these similar ventilation was 2.4 and 7.3 ml/l/min respectively. The difference between these two calculations would be 1.078 l of O_2/min at a ventilation of 220 l/min which is double the reported measurement error ($\pm$ 0.513 l/min). Factors that can influence measurements of $\dot{V}O_2$resp are : lung volume, breathing pattern, posture, and whether the breathing pattern is voluntary.

When the same level of ventilation is performed at a lung volume greater than functional residual capacity (FRC), the $\dot{V}O_2$resp is greater even when the work rate and the pressure time product are controlled (10). The possible reasons Collett and Engel(10) offer for the increased VO_2resp at high lung volumes are: 1. less mechanical efficiency at higher lung volumes due to a shorter initial muscle length; 2. greater recruitment of accessory muscles or postural muscles; 3. more unmeasured work due to greater chest wall distortion at higher lung volumes. Klas and Dempsey (19) demonstrated that during voluntary ventilation (MVV test), the lung volume is greater than during the involuntary ventilation of high exercise. Therefore, the $\dot{V}O_2$resp during testing is likely to be greater for the same level of ventilation during exercise.

Both breathing frequency and tidal volume affect the level of ventilation that can be achieved and also the energy cost of ventilation. The optimal breathing frequency to obtain the highest ventilation, was suggested by Shephard (41) to be 100 breaths per minute (bpm) while 30-60 bpm was suggested by Otis (32) along with a tidal volume that is not greater than a subjects' inspiratory capacity. Ogilvie et al (29) reported that 24 normal subjects achieved higher ventilation when they spontaneously selected a breathing frequency of 93 bpm than when they were instructed to breathe with the same frequency.

The number of muscles that are active during breathing tests also influences the $\dot{V}O_2$resp. It is not possible to totally isolate the RM during breathing tests since the following muscles are attached to the rib cage and yet are not considered accessories

to breathing: serratus anterior, pectoralis major and minor, and the latissimus dorsi. As part of the protocol of measuring the $\dot{V}O_2$resp by altering the resistance of the inspired air, McCool et al (26) suspended their subjects' arms by rubber tubing and also instructed the subjects not to contract their leg muscles in order to limit the use of these other muscles during the breathing tests. To reduce the experimental error, body position should be standardized between subjects during studies where $\dot{V}O_2$resp is measured for inter or intra-subject comparison.

Otis (32) also points out that VO_2resp measurements can only be accurate if a "steady state of gas exchange is rigorously maintained during the period of study." This criterion would limit the accurate measurement of $\dot{V}O_2$resp to tests of at least a three minute duration and then only those with a set breathing frequency and lung volume.

In spite of a protocol with a standardized breathing pattern, intra-subject variability can be quite large. Collett and Engel (10) measured the $\dot{V}O_2$resp of four subjects at different tidal volumes for the same level of ventilation. The protocol required the subjects to maintain a constant duty cycle (Ti/TT), inspiratory flow, inspiratory resistance, tidal volume and end expiratory lung volume. However, the $\dot{V}O_2$resp of one subject ranged from 30 to 97 ml/min at the same work rate of 65 J/min.

Bradley and Leith (4) reported an increase in respiratory muscle endurance of 19% and an associated increase in $\dot{V}O_2$resp of 67% in four subjects following 5 weeks of normocapnic hyperpnea training. Their conclusion, based on these results, was that the aerobic endurance capacity of the respiratory muscles had changed as a result of the

hyperpnea training program. Since the measurements of $\dot{V}O_2$resp were not made at either the same ventilation or test duration, both before and after training, a more accurate conclusion might have been that the higher sustained ventilatory capacity following training was associated with a higher $\dot{V}O_2$resp.

FATIGUE

There are two techniques commonly used to create ventilatory muscle fatigue: either volume overloads or resistance overloads. The effects of manipulating either of these parameters can be measured directly with EMG or thoracic pressure studies, or measured indirectly by RM strength and endurance. As RM fatigue, the high/low power frequency ratio as measured by EMG decreases, and RM strength and endurance decrease so that the muscles are unable to maintain a specified force of contraction.

Direct assessment of respiratory muscle fatigue is complicated by the location of the muscles as most of the respiratory muscles are inaccessible to surface examination. For the same reason, the length of the muscle fibers is difficult to determine and knowing the length of the muscle fibers is useful for standardizing protocols, especially since the length of the respiratory muscles change as the lung volume change and muscle fibers fatigue at different rates at different lengths. The abdominal muscles and even the abdominal contents can influence the length of the diaphragm. Celli (8) describes the action of the diaphragm as it "leans" against the abdominal contents. If the abdominal contents are rigid, they act as a fulcrum to lift and expand the rib cage;

if the contents are compliant, the synergic effect would be lessened. These factors should be considered when designing a research protocol studying RM fatigue and involving inter and intra-subject variability.

In the following section, the first five studies report on respiratory muscle fatigue as a result of artificial (laboratory induced) hyperpnea and the next three studies report on respiratory muscle fatigue following total body exercise.

In 1970, Freedman (14) published the results of an experiment to determine the maximum ventilation that could be maintained for 4 minutes. The results obtained with 20 healthy subjects (age 30 $\pm$ 7.6 yr) showed an inverse relationship between the level of ventilation and the time it could be sustained. The subjects were able to maintain 80% of their MVV_{15} for one minute, 75% for 2 minutes and 72% for 4 minutes and Freedman estimated that 50% could be maintained indefinitely. Freedman excluded the influence of mechanical factors and reached the conclusion that the level of ventilation that could be maintained during the 4 minute MVV was limited by the power of the RM and possibly by the subjects' discomfort.

In 1979, Roussos et al (37) conducted an experiment to assess inspiratory muscle fatigue while thoracic pressure and volume changes were measured. Transdiaphragmatic pressure (Pdi) was used to maintain constant lung volumes and gastric pressure (Pga), esophageal pressure (Pes) mouth pressure (Pm) and rib cage and abdominal motion were monitored to assess muscle recruitment. Five normal subjects breathed against increasing resistances for prolonged periods of time at two

different lung volumes: functional residual capacity (FRC) and at FRC plus one half inspiratory capacity (FRC + 1/2 IC). The results of the study showed that respiratory muscle endurance was inversely related to the mouth pressures required to overcome a resistance and that the endurance for a similar pressure was reduced at the higher lung volume. Also, both the mouth pressure that could be maintained indefinitely (critical mouth pressure) and the maximal mouth pressure were lower at FRC + 1/2 IC than at FRC (33% and 80 cmH_2O vs 60% and 130 cmH_2O). As a result of monitoring the Pdi and Pga pressure changes, Roussos et al concluded that as the diaphragm fatigued, other muscles were recruited. For example, they describe a minimal Pdi with a decrease in abdominal pressure below resting end-expiratory value as an indication of the recruitment of intercostal muscles during inspiration.

Bai et al (1) conducted a study similar to Freedman's (14) but monitored the diaphragmatic EMG, Pdi and pressure-frequency curves concomitant with hyperpnea. The five healthy subjects (age 31 $\pm$ 3.3 yr) were able to maintain 86% of their MVV_{12} for less than 3 minutes, 79% for 6 minutes and 76% for 20 minutes. Following hyperpnea at 79% and 76% of MVV, both maximal inspiratory and expiratory pressures and the MVV_{12} were decreased. Diaphragmatic fatigue, as measured by EMG, is defined as an increase in the amplitude of the low frequency components (20-40 Hz) and a decrease in the high frequency components (130-238 Hz) (15). Due to a decreased high/low frequency ratio of the diaphragmatic EMG, decreased Pdi_{max} and

decreased pressure frequency curves, Bai et al concluded that the decreased respiratory muscle strength and sprint measures were a result of diaphragmatic fatigue.

Martin et al (24) investigated the effects of 150 minutes of isocapnic hyperventilation on the short term maximal running performance of 9 healthy subjects (age 26 $\pm$ 3 yr). The subjects were able to maintain 66% (119 l/min) of their MVV_{12} for 150 minutes and the result was a decrease in maximal running time to 6.5 minutes compared to the control running time of 7.6 minutes. Exercise ventilation were decreased as well, from 124 l/min during the control run to 117 l/min following hyperventilation. The maximum heart rate and $\dot{V}O_2max$ also decreased which may have indicated less effort on the part of the subjects. However, the heart rates from both sessions (187 and 179 beats/minute) are greater than 90% of the predicted maximum heart rate (176 beats/min) and also, the respiratory exchange ratio (RER) was greater than 1.15 for both exercise sessions and these parameters indicate maximal effort during the exercise tests. The authors compared the decline in ventilation and oxygen consumption of their subjects during the 150 minutes of hyperventilation with similar studies using leg exercise, and speculated that the main cause of fatigue in the respiratory muscles would be depleted glycogen stores in a manner similar to that reported for the leg muscles.

The sternomastoid (SM) muscle is an accessory muscle activated at high lung volumes and also during increased ventilatory demands such as in exercise. Moxham et al (28) developed a technique to measure the force-frequency curves of the SM and

recorded evidence of substantial low frequency fatigue following resistance and hyperpnea challenges. One part of the study required four healthy subjects to breathe for 200 breaths against a resistance that required 70% of their maximum inspiratory strength. The second part of the study required three subjects to perform an MSVC test for 10 minutes and the mean ventilation sustained was 59% of the MVV_{15}. Frequency-force curves were determined both 10 minutes before and 10 minutes after each of these breathing exercises. The post exercise frequency-force curves were shifted to the left with the higher frequencies showing normal force output but the force at the low frequencies was reduced. An EMG was also recorded for two of the subjects and the results of a smoothed rectified EMG showed that following exercise, a higher amplitude was produced to generate the same force as before the inspiratory loading. Moxham et al found the EMG response of the SM to be very similar to the response of other skeletal muscles, namely the quadriceps and adductor pollicis, when fatigue was produced and measured by the same technique.

The next three studies discuss the effects of whole body exercise on the respiratory muscles in contrast to the previous studies that reported the effects of artificial hyperventilation on the respiratory muscles.

Loke et al (21) measured the respiratory muscle strength and sprint performance of four healthy males (age 35 $\pm$ 3.4 yr) before and after a marathon run. The mean running time was 3:24 hours and the estimated ventilation during the run was 75 l/min. Following the run, each of the subjects demonstrated a decreased maximal inspiratory

pressure (138.5 $\pm$ 7.6 cmH_2O vs 165.8 $\pm$ 11), decreased maximal expiratory pressure (173.0 $\pm$ 22.6 cmH_2O vs 240.0 $\pm$ 20.4), decreased transdiaphragmatic pressure during inspiration (Pdi_{IC}) (63.3 $\pm$ 7.0 cmH_2O vs 78.8 $\pm$ 11.6, and decreased MVV_{15} (161.2 $\pm$ 23.2 l/min vs 178 $\pm$ 24.2). The authors interpreted the decreases in respiratory muscle strength and sprint measurements as an indication of fatigue of the respiratory muscles.

The effects of 60 minutes of exhaustive running was studied in 8 runners and 8 non-runners (age 25 $\pm$ 4 yr) by Bender and Martin (3). The measure of respiratory muscle endurance they used was a 60 second MVV with a set breathing frequency of 60 breaths per minute. The MVV_{60} was performed before and after a 60 minute treadmill run and the ventilation decreased significantly in all subjects (155 to 140 l/min) with the non-runners showing the greatest change. The non-runners' MVV_{60} decreased from 158.7 $\pm$ 22 to approximately 141.0 l/min. The runners' MVV_{60} decreased from 150 $\pm$ 23 l/min pre exercise to approximately 140 l/min post exercise. The same procedure was performed with 8 subjects and a shorter duration of exhaustive exercise (less than 10 minutes) and the results did not show a decrease in the MVV_{60}. Bender and Martin concluded that ventilatory endurance may affect exercise ventilation and they speculate that the cause of the decreased endurance is depletion of glycogen stores or inefficiency of breathing during exercise. Limiting the breathing frequency during the MVV_{60} may have influenced the results of this study. The values of 158 and 151 l/min of the non-runners and runners is precisely what would be predicted if a tidal volume of 50% of the vital capacity (5.11 $\pm$ 0.5 l) is assumed.

Selecting any breathing frequency does not take into consideration that the speed of muscle contraction may vary between individuals and that a small vital capacity may not limit a person's MVV. Perhaps it would be more appropriate to determine each subject's breathing frequency during exercise and specify that as their target frequency.

Diaphragmatic EMG and thoracic pressures were used by Bye et al (6) to directly measure respiratory muscle fatigue on 7 subjects (age 26-36 yr) following exhaustive exercise at 80% of their maximal power output. The average exercise time was 6 minutes, ventilation was 122 $\pm$ 21 l/minute and the $\dot{V}O_2$ was 4.2 $\pm$ 1 l/min. A decreased High/Low frequency of the diaphragm of 20% was recorded as well as a drop in Pdi_{max} from 190 $\pm$ 26 to 167 $\pm$ 24 cmH_2O. Both of these responses signify diaphragmatic fatigue following short term exercise. These results are in contrast to those of Bender and Martin (3).

These studies demonstrate that the RM of healthy people are fatiguable and perhaps may contribute to decreased ventilation which may result in decreased aerobic performance in highly trained athletes.

TRAINING

Athletes involved in sports requiring a high degree of aerobic fitness have greater respiratory muscle endurance than non-athletes as a result of the high levels of ventilation they maintain during their cardiovascular workouts. Martin and Stager (25) compared the respiratory muscle endurance of 8 endurance athletes (age 19 $\pm$ 2.8 yr) with 8 non-athletes (age 22 $\pm$ 2.8 yr). The short-term maximal ventilation (MVV_{12})

was similar for athletes and non-athletes (157 vs. 146 l/min), however, the time the athletes could maintain 80% of the MVV_{12} was significantly longer (11 $\pm$ 5.7 vs. 3 $\pm$ 2.8 min) and the level of $\dot{V}_E$max they could reach during a progressive ventilation test where the target ventilation is increased every two minutes, was higher (118 $\pm$ 17 vs. 95 $\pm$ 8.5 l/min). To determine if the greater respiratory muscle endurance of the athletes was a result of genetic predisposition or cardiovascular training, Martin and Chen (23) used the same protocol as Martin and Stager (25) to compare 8 young distance runners with 8 of their untrained siblings. The athletes were larger than their siblings but both VC and FEV_1 were normal for each group. The athletes MVV_{12} was greater (172 $\pm$ 31 vs. 107 $\pm$ 25 l/min) but when these values were corrected for VC, the two groups were not different. The athletes, however, did maintain 80% of their MVV_{12} longer than their siblings (7 vs. 3 min) and the athletes reached 75% of their MVV_{12} on a progressive ventilation test while the siblings reached only 62%. Both of these studies demonstrate greater respiratory muscle endurance of athletes and the results of Marten and Chen suggest that aerobic training is responsible.

Respiratory muscles are skeletal muscles and subject to the same adaptive responses to specific stimuli as other skeletal muscle. Pardy and Leith (33) list the training regimens for specific adaptive responses as follows: strength requires high-load, low-repetition; for endurance, low-load with high-repetitions; speed, rapid high-repetition; length training, the muscle is to be trained above or below it's resting length. The following studies are examples of training the respiratory muscles for improving

either strength or endurance in healthy subjects. To date, there is little information on training RM for speed or optimal length.

In 1976, Leith and Bradley (20) published the results of five weeks of either respiratory muscle strength or endurance training and compared the results to a non-training control group. There were 4 healthy subjects in each group whose mean age was 31.3 $\pm$ 5.8 yr. Strength training consisted of five, 1/2 hour sessions per week and subjects performed maximal inspiratory and expiratory maneuvers at different lung volumes (the range was 20% intervals of their vital capacity). The apparatus necessary for this training was a spirometer and a solenoid-operated valve. Following training, the maximum inspiratory pressure increased 47% from 110 $\pm$ 32 to 162 $\pm$ 22 cmH_2O. The endurance training protocol consisted of ventilation that could be sustained for 0.5 to 15 minutes with a normocapnic atmosphere maintained by partial rebreathing. Compressed air was supplied to the breathing circuits at different flows and the subjects were required to keep the 'target' bag empty. The duration of each breathing session was inversely related to the amount of air supplied with 15 minutes as the maximum duration and a total or 19-25 minutes of hyperpnea per session. Leith and Bradley define their measure of endurance, the sustained ventilatory capacity (SVC), as the asymptote of the curve of the 8-10 selected maximum points of ventilation vs. time the subjects could maintain. The SVC increased from 163 $\pm$ 45 to 197 $\pm$ 69 l/min after five weeks of training. The MVV_{15} increased as well by about 14% and the authors

reported the lung volume and inspiratory flow of one subject showed the performance of the MVV was at a higher lung volume and greater flow than before training.

Keens et al (18) compared the results of 4 weeks of normocapnic hyperpnea training between 4 subjects (age= 28.3 $\pm$ 2.6 years) and 7 non-training controls (age 31.9 $\pm$ 5.3 yr). The training regimen was 25 minutes per day, 5 days per week with apparatus similar to that described by Leith and Bradley (20). Subjects were allowed to select their own posture and breathing pattern during the testing and training sessions. The muscle training group increased the MSVC from 134 $\pm$ 36 to 163 $\pm$ 42 l/min while the control group did not change (110 $\pm$ 25 l/min). In addition to the study of ventilatory muscle endurance training, Keens et al present the MSVC divided by the FEV_1 as a correction factor to permit inter-subject comparisons. The $MSVC/FEV_1$ value was the same for 11 healthy subjects with normal spirometry compared to 11 subjects with cystic fibrosis with abnormal spirometry. The $MSVC/FEV_1$ for the 4 training subjects increased from 34.5 $\pm$ 3.2 to 42.9 $\pm$ 5.8 following training.

Morgan et al (27) examined the effects of 3 weeks of respiratory muscle endurance training on ventilatory endurance, $\dot{V}O_2max$ and cycling performance of moderately trained cyclists. Comparisons were made between the 4 experimental subjects (age= 24 $\pm$ 2 yrs.; $\dot{V}O_2max$ 50.7 $\pm$ 4 ml/kg/min) and 5 control subjects (age= 25 $\pm$ 2 yrs.; $\dot{V}O_2max$ 50.2 $\pm$ 4 ml/kg/1). The apparatus for the training included a vacuum cleaner motor to supply flow with a variable output generator to control the rate.

Normocapnic hyperpnea was maintained by rebreathing a portion of expired CO_2 and subjects watched the spirometer bell as a target for ventilation levels. There were 5 training sessions per week for 3 weeks and subjects breathed at 85% of their MVV_{15} for a maximum training time of 28 min per day. The MVV_{15} was measured each week and the training ventilation was adjusted to 85% of this new value. Before and after training, each subject performed the MVV_{15} and the measure of breathing endurance was the amount of time a subject could breathe at 100% MVV. In addition to the ventilation tests, VO_2max was also determined by an incremental cycle ergometer test with expired gas analysis for metabolic data. As a measure of aerobic endurance performance, cycling tests at 95% of the VO_2max tests were included. Following the training period, both the MVV_{15} and the endurance breathing test increased for the experimental subjects (214 $\pm$ 16 to 243 $\pm$ 28 l/min; 48 $\pm$ 20 to 804 $\pm$ 188 seconds respectively) with no other test differences. There is no doubt that both of these variables increased but the intensity of the training stimulus is difficult to assess. The target ventilation for the first week of the study should have been 181 l/min (85% of MVV_{15}) and yet the reported average ventilation was 165 l/min for this week and 190 l/min reported only for week 3. The length of each training bout was to be determined by the length of time the target ventilation could be maintained and when the subjects' ventilation dropped below the target ventilation the training bout was ended. The authors assume the MVV_{15} increased as a result of increased strength but did not make any strength measurements.

Belman and Gaesser (2) used normocapnic hyperpnea to train 12 elderly subjects (age = 67 $\pm$ 3 yr; $\dot{V}O_2$max = 23.8 $\pm$ 5 ml/kg/min) for a period of 8 weeks. There were 4 sessions per week with 30 minutes of hyperpnea training per session. Before and after training, the following tests were conducted on the experimental group and on an age matched control group (n=13): 10 minute MSVC, MVV_{12}, vital capacity (VC), FEV_1, $\dot{V}O_2$max and a steady state cycle test at 70% of the ventilation achieved during the $\dot{V}O_2$ max. For the experimental group, the MSVC increased (72.9 $\pm$ 26 to 86.9 $\pm$ 20 l/min), the MVV_{12} increased (115.5 $\pm$ 40 to 135.5 $\pm$ 35.7 l/min) and the VC increased (3.35 $\pm$ 1 to 3.5 $\pm$ 1 liter) and no changes occurred with the control group. The incremental and steady state exercise test results were similar to the study of MOrgan et al (27) with no changes in either the metabolic or ventilatory parameters.

To determine the effects of running on ventilatory muscle function, Robinson and Kjeldgaard (36) tested 11 normal subjects (age= 36.7 yr) following 20 weeks of supervised exercise and compared their results to a control group (age = 26.8 yr) that did not participate in the training. The experimental group participated in an exercise program of running at 80% of their maximum predicted heart rate for 40 minutes a day, three times a week. The MSVC of the training group increased by 15.8% (88 $\pm$ 20 l/min to 94 $\pm$ 18 l/min), the MVV_{12} increased 13.6% (127 $\pm$ 15 l/min to 144 $\pm$ 17.8 l/min) and the maximal expiratory pressure at the mouth, increased from 141 $\pm$ 60 cmH_2O to 155.5 cmH_2O) while the same measurements for the control subjects did not

change. The authors concluded that this running program offered a sufficient stimulus to increase both ventilatory muscle strength and endurance.

The results of these studies suggest that it is possible to train the endurance and the strength of the respiratory muscles in healthy subjects whose ages range from 24 to 67 years, whose fitness levels range from sedentary to moderately fit and by either artificial methods of high volume overload or by a supervised running program.

APPENDIX B: Anthropometric Data

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Subject		Height (cm)	Weight (kg)	Age (yr)	FVC (l)	FEV_1 (l)
Pre-training						
	1	165	63.0	26	4.88	4.19
	2	171	68.8	20	5.12	4.50
	3	171	63.5	25	5.00	4.42
	4	175	71.0	20	5.27	4.19
	5	188	80.0	20	7.09	4.82
	6	178	73.4	20	5.76	4.65
	7	185	85.0	24	6.73	5.86
	8	175	74.7	29	5.75	4.51
	9	175	69.6	23	5.17	4.52
	10	176	64.4	17	5.03	4.59
Post-training						
	1		61.5	26	4.65	3.82
	2		68.5	20	5.42	4.79
	3		63.0	26	5.33	4.55
	4		71.7	20	5.53	4.44
	5		78.5	20	7.32	4.75
	6		72.2	20	5.80	4.46
	7		83.0	24	6.74	5.83
	8		74.5	29	5.35	4.15
	9		64.9	23	5.18	4.67
	10		65.0	17	4.80	4.42

APPENDIX C: Maximal Exercise Data

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Subject	$\dot{V}O_2max$ (ml)	$\dot{V}O_2$ (ml·kg·min)	RER %	$\dot{V}_Emax$ (l/min)	HR (bpm)	$\dot{W}max$ (watts)
Pre-training						
1	4017	63.9	1.31	162.9	184	349
2	4575	66.0	1.34	191.3	198	415
3	4089	63.6	1.32	150.9	180	380
4	4641	66.0	1.39	175.0	176	415
5	4919	61.5	1.52	204.9	192	415
6	4842	66.0	1.23	190.9	197	360
7	5134	60.4	1.48	224.6	198	455
8	5690	76.2	1.04	143.5	188	390
9	5102	73.3	1.11	153.0	194	400
10	4127	64.1	1.29	145.0	195	330
Post-training						
1	4142	66.7	1.37	171.9	180	362
2	4765	70.2	1.28	191.3	198	455
3	4380	70.1	1.23	159.3	174	390
4	4481	63.0	1.35	176.7	178	400
5	4598	59.0	1.35	186.4	188	330
6	4855	67.3	1.16	187.1	194	360
7	4943	60.0	1.22	187.0	196	445
8	5558	74.6	1.03	135.5	180	390
9	4585	70.6	1.03	160.5	193	390
10	4084	63.8	1.30	167.4	187	366

APPENDIX D: Performance Cycle, MVV and MSVC Data

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Subject	Tlim (seconds)	$V_E$tlim (l/min)	MVV (l/min)	MSVC (l/min)
Pre-training				
1	351	165.2	229.5	145.2
2	343	193.1	202.1	170.3
3	459	161.9	206.0	157.1
4	298	165.7	202.2	143.5
5	260	194.2	187.5	160.7
6	360	183.6	219.5	159.8
7	228	196.9	247.2	194.2
8	380	200.0	209.3	153.0
9	225	159.0	194.3	145.5
10	450	130.4	205.8	123.2
Post-training				
1	449	164.5	202.8	163.7
2	382	181.2	241.7	192.1
3	801	163.2	230.2	172.4
4	284	173.2	208.8	164.3
5	223	210.4	198.0	176.9
6	299	190.3	224.9	156.6
7	384	216.3	251.5	202.5
8	260	165.2	.	127.2
9	309	158.1	227.5	147.0
10	460	151.3	189.2	114.4

APPENDIX E: Breathing Patterns for Ventilation Tests

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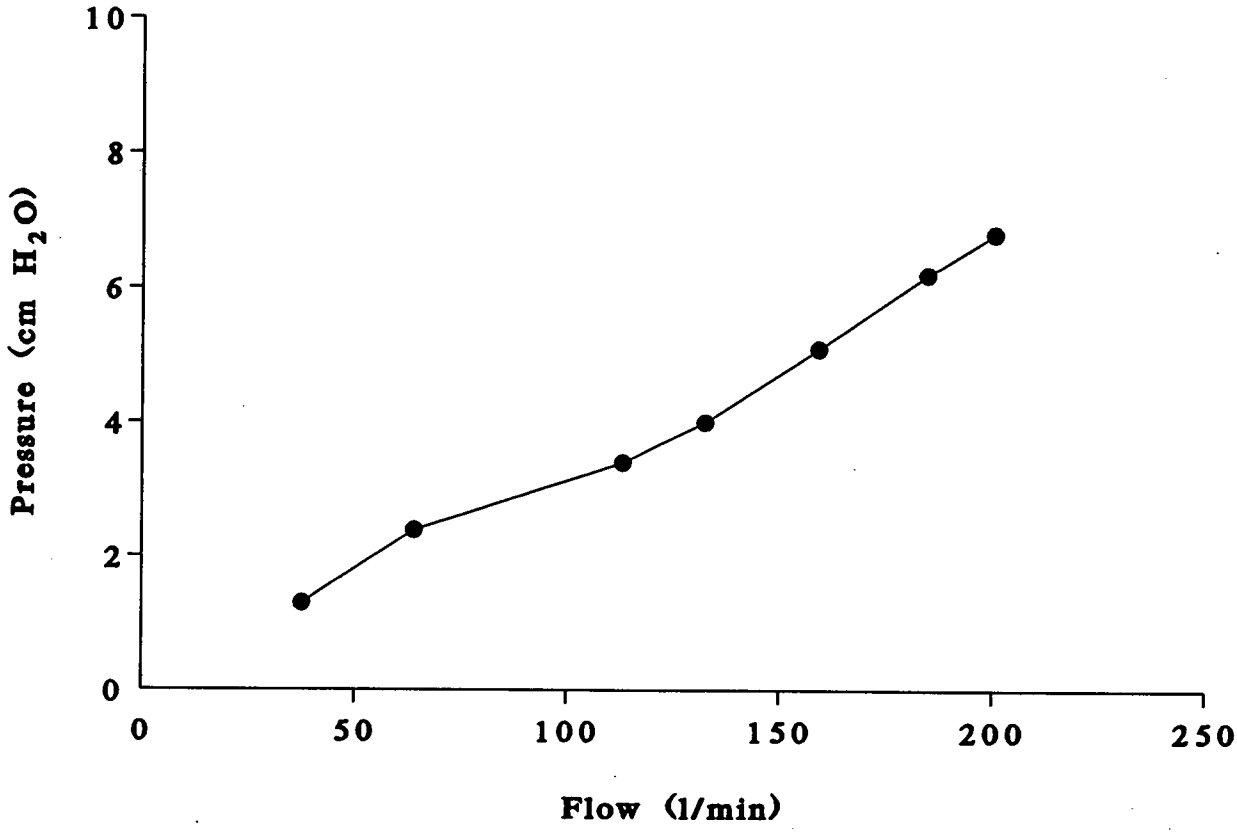
Subject	MSVC (l/min)	V_T (l)	RR (bpm)	MVV (l/min)	V_T (l)	RR (bpm)
Pre-training						
1	145.2	2.27	65	229.5	1.48	155
2	170.3	2.76	63	202.1	1.22	165
3	157.1	2.48	64	206.0	1.87	110
4	143.5	2.50	59	202.2	2.37	85
5	160.7	2.80	58	187.5	1.63	115
6	159.8	2.84	57	219.5	2.19	100
7	194.2	3.63	58	247.2	2.47	100
8	153.0	2.48	62	209.3	2.20	95
9	145.5	2.14	69	194.3	1.85	105
10	123.2	1.99	63	205.8	2.42	85
Post-training						
1	163.7	3.14	55	202.8	1.93	105
2	192.1	3.60	55	241.7	2.19	110
3	172.4	3.22	55	230.2	2.19	105
4	164.3	3.63	48	208.8	2.45	85
5	176.9	3.33	54	198.0	2.08	95
6	156.6	2.32	54	224.9	1.50	150
7	202.5	3.80	54	251.5	3.35	75
8	127.2	2.16	59			
9	147.0	2.25	67	227.5	1.98	115
10	114.4	1.96	59	189.2	1.72	110

APPENDIX E: Breathing Patterns (cont'd)

Subject	$\dot{V}_E$max (l/min)	V_T (l)	RR (bpm)	$\dot{V}_E$tlim (l/min)	V_T (l)	RR (bpm)
Pre-training						
1	162.9	3.07	53	165.2	2.81	59
2	191.3	3.13	61	193.1	2.85	68
3	151.0	2.90	52	161.9	3.10	53
4	175.0	3.50	50	165.7	3.26	51
5	204.9	3.98	52	194.2	3.30	60
6	190.9	3.51	55	183.6	3.09	57
7				196.9	4.10	48
8	143.5	2.77	52	181.3	3.09	35
9	159.0	2.74	59	130.4	3.08	42
10	145.0	2.64	55	130.4	2.59	52
Post-training						
1	171.9	2.75	63	147.9	3.02	54
2	191.3	3.30	57	181.2	3.10	58
3	159.3	2.90	55	163.2	2.98	55
4	176.7	3.55	50	173.2	3.55	49
5	186.4	4.26	44	210.4	3.90	54
6	187.1	4.24	45	190.3	2.98	64
7	187.0	3.84	49	196.3	4.21	47
8	133.5	3.54	38	166.5	3.20	52
9	160.6	2.34	69	158.1	2.97	53
10	151.3	2.62	58	167.4	2.58	66

APPENDIX F: Resistance of the Ventilatory Endurance Apparatus



Appendix F: Resistance of the Ventilatory Endurance Apparatus.
As air flow increases, resistance increases.