THE EFFECT OF A HIGH INTENSITY BOUT OF EXERCISE ON MAXIMUM EXPIRATORY PRESSURE IN HIGHLY TRAINED INDIVIDUALS

by

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(ABSTRACT)

Ten well trained cyclists were studied and compared with 12 untrained subjects from a previous study to determine the effects of a high intensity, constant workload bout of cycling on maximum expiratory pressure (Pe_{max}). Subjects completed a graded exercise test on a Monark cycle ergometer while expired gases were collected to determine maximal oxygen consumption (VO_{2max}). Subjects then returned on a second day when measurements of each subject's Pe_{max} were made prior to riding at the workload corresponding to 90% of their VO_{2max} until exhaustion. Measurements of expiratory pressure (Pe) were then made immediately post exercise (Pe_{IPE}), one minute post exercise (Pe_{1MIN}), three minutes post exercise (Pe_{3MIN}), and five minutes post exercise (Pe_{5MIN}). Trained cyclists had a significantly higher Pe_{max} ($x = 116.43 \pm 7.76$ mmHg) than did untrained subjects ($x = 65.75 \pm 7.09$ mmHg). Also trained cyclists generated a higher absolute Pe throughout recovery than did the untrained subjects. Although expiratory pressure decreased after exercise in both groups, the relative change in Pe over the recovery period, expressed as a percentage of Pe_{max}, was not different between trained and untrained. Pe_{IPE} was decreased to 81.87% ± 3.12 of Pe_{max} in trained subjects and 82.35% ± 2.85 in untrained subjects ($p < .05$), recovering somewhat at 1 minute to 89.19% ± 3.59 of Pe_{max} in trained and to 87.74% ± 3.27 in untrained ($p < .05$) but did not recover to resting levels in either group. Pe_{3MIN} and Pe_{5MIN} remained at the same level as Pe_{1MIN} in both groups. Therefore, a high intensity, short term exercise bout caused expiratory pressure to be decreased in both trained and untrained subjects.

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Chapter I

INTRODUCTION

Most of the research in the area of respiratory muscle fatigue has concentrated on inspiratory muscle fatigue. During exercise, however, the expiratory muscles also play an important part by providing the quick, forced exhalation needed to increase ventilation rates to levels needed to maintain aerobic exercise. Expiratory muscle fatigue, therefore, is of extreme interest in the study of the limits of the respiratory system in exercise. The research in the area of inspiratory and expiratory muscle fatigue, however, has been limited.

Coast, Clifford, Henrich, Stray-Gundersen & Johnson (1990) found that untrained subjects acquire inspiratory muscle fatigue after a relatively short period of exercise. After an incremental cycling VO_{2max} test to exhaustion, untrained subjects showed a decrease in maximum inspiratory pressure (Pi_{max}) which is a measure of inspiratory muscle strength. Trained subjects did not, however, show any decreases in Pi_{max} following the same incremental exercise test. Endurance athletes have been shown to display greater ventilatory endurance than non-athletes (Martin & Stager, 1981). The differences between trained and untrained subjects may be due to a training effect or it may indicate a genetic predisposition for stronger, more fatigue resistant respiratory systems in people who choose to train.

Biochemical and histochemical changes in certain respiratory muscles can also be elicited by endurance training. Farkas & Roussos (1984) found that endurance training by running in emphysematous hamsters provided some protection against respiratory muscle fatigue by preserving muscle fibers of the intercostals. Moore & Gollnick (1982) examined the muscle fibers of rat diaphragm and intercostal muscles after training by treadmill running. They found succinate dehydrogenase (SDH), which is used to estimate oxidative potential of a muscle, increased in the diaphragm. But they found no change in SDH in the intercostals. Likewise, Uribe, Stump, Tipton & Fregosi (1992) found that endurance training in rats led to increases in citrate synthase (CS), also a measure of oxidative capacity, in locomotor muscles and the diaphragm but not in the abdominal expiratory muscles. Thus, cellular changes in the respiratory muscles may not occur uniformly.

Suzuki, Suzuki & Okubo (1991) reported a decrease in maximum expiratory pressure (Pe_{max}), which is a measure of expiratory muscles strength, for up to 60 minutes following expiratory resistive loaded breathing. But, expiratory resistance does not come into play normally during exercise. Rather, during exercise, the stressor on the respiratory muscles is the high ventilation rates. Loke, Mahler & Virgulto (1984) investigated the effects of long duration exercise on Pe_{max}, Pi_{max} and maximum voluntary ventilation (MVV), which is a measure of ventilatory endurance. They found decreases in all measures following a marathon race indicating that a long term exercise bout may cause both inspiratory and expiratory muscle fatigue in highly trained runners, possibly due to the high ventilation rates and energy depletion. Likewise, Mahler & Loke (1981) found a decrease in forced vital capacity (FVC) in 15 runners following an ultramarathon which may indicate some respiratory muscle fatigue. Although the reduction in FVC could be a result of airway obstruction, the researchers feel that respiratory muscle fatigue is to blame because the FEV_1 / FVC ratio remained constant after the race ruling out large but not small airway obstruction. Unfortunately, no direct measures of respiratory muscle function, such as respiratory pressures, were made. Thus, it can not be known definitively whether respiratory muscle fatigue occurred in this study.

In a study similar to the present study, Bye, Esau, Walley, Macklem & Pardy (1984) studied Pe_{max} and diaphragmatic EMG in untrained subjects before and after an exercise bout at 80% of maximum workload attained during a graded exercise test. The subjects performed the study in both 40% O_2 and in room air. Pe_{max} did not change after the short term, high intensity exercise bout, however, diaphragmatic EMG, did predict fatigue using a 20% fall in the high-to-low ratio as the criterion. Whereas Bye, et al. used an bout of exercise at 80% of VO_{2max}, Wilkins (1991) performed a study in this lab, on which the current study is based, showing expiratory muscle fatigue in untrained subjects, as indicated by a drop in Pe_{max} in untrained subjects after a high intensity cycling bout at 90% of VO_{2max}. Therefore, it is of interest to determine if expiratory muscle fatigue will be detected in trained cyclists after an endurance ride at 90% of VO_{2max}.

Statement of the Problem

The research on respiratory muscle fatigue and exercise is limited and conflicting. Expiratory muscle fatigue has been detected, but only after long term exercise, such as a marathon. The purpose of this study is to determine whether a short term, high intensity exercise bout at 90% of VO_{2max} will cause significant changes in Pe_{max} in trained cyclists up to 5 minutes post exercise. If expiratory muscle fatigue is detected, comparing these findings to previous findings in untrained subjects will indicate whether trained individuals may possess some resistance to fatigue of the respiratory muscles. The absolute expiratory pressure maximums of trained and untrained subjects will also be compared. Finally, the relative changes in expiratory pressure, expressed as Pe / Pe_{max} at IPE, 1 minute, 3 minutes, and 5 minutes post exercise, will be compared between trained and untrained subjects to investigate the differences in respiratory muscle fatiguability.

ResearchHypotheses

The following null hypotheses were developed to define the objectives of the present study:

Ho_1: There is no difference between Pe_{max} and Pe_{IPE}, Pe_{1MIN}, Pe_{3MIN}, or Pe_{5MIN} following an exhaustive bout of cycling at 90% of VO_{2max} in trained cyclists.

Ho_2: There is no difference between Pe_{max}, Pe_{IPE}, Pe_{1MIN}, $Pe3_{MIN}$ or Pe_{5MIN} in trained subjects and untrained subjects.

Ho_3: There is no difference between Pe/Pe_{max} in trained and untrained subjects during recovery from an exercise bout at 90% of VO_{2max}.

Significance of the Study

The purpose of this study is to investigate the maximum expiratory pressures generated by highly trained individuals before and after a high intensity, exhaustive bout of exercise. The study will examine changes in expiratory pressure development, which is a reliable indicator of changes in expiratory muscle performance after high levels of work (Rochester & Braun, 1978). Results of this study will provide more information on the limits and fatiguability of the expiratory muscles.

Previous studies have indicated that long term, exhaustive exercise can fatigue inspiratory and expiratory muscles (Loke, et al., 1982). Also, high intensity, short term exercise in untrained subjects can induce inspiratory muscle fatigue (Bye, et al., 1984). Likewise, high intensity, short term exercise can cause expiratory muscle fatigue in untrained subjects (Wilkins, 1991). This study will expand on that by investigating the effects of high intensity, short term exercise on expiratory muscle function in highly trained subjects.

Delimitations

The following delimitations were incorporated into the study by the investigator:

1. The subject population was delimited to 7 male and 3 female trained cyclists ranging in age from 20 to 32.
2. The investigation was delimited to include only those subjects who have been cycling at least 100 miles per week for the last 3 months.
3. The investigation was delimited to apparently healthy individuals with no known respiratory disorders who did not use any tobacco products.
4. The evaluation of expiratory muscle performance in this study was delimited to the measurement of forced expiratory pressure at IPE, 1 minute, 3 minutes, and 5 minutes.
5. This evaluation was delimited to the muscles of expiration.

Limitations

The investigator acknowledged the following limitations:

1. Due to the specific population evaluated in this study, the results are limited to trained cyclists.

2. The results of this study are limited to the ability of the expiratory muscles to generate maximum pressure.

Definitions and symbols

1. Forced expiratory volume ($FEV_{1.0}$) - The volume of air that can be forcibly exhaled in the first second after a maximal inhalation.
2. Maximum expiratory pressure (Pe_{max}) - The maximum amount of pressure that can be generated after a maximal inhalation.
3. Expiratory pressure immediately post exercise (Pe_{IPE}) - The maximum amount of pressure that can be generated after a maximal inhalation measured within 20 seconds of termination of the exercise bout.
4. Expiratory pressure at 1 minute post exercise (Pe_{1MIN}) - The maximum amount of pressure that can be generated after a maximal inhalation measured at 1 minute after termination of the exercise bout.
5. Expiratory pressure at 3 minutes post exercise (Pe_{3MIN}) - The maximum amount of pressure that can be generated after a maximal inhalation measured at 3 minutes after termination of the exercise bout.
6. Expiratory pressure at 5 minutes post exercise (Pe_{5MIN}) - The maximum amount of pressure that can be generated after a maximal inhalation measured at 5 minutes after termination of the exercise.
7. Maximum inspiratory pressure (Pi_{max}) - The maximum amount of pressure that can be generated after a maximal exhalation.
8. Tidal volume - The volume of gas inspired or expired during each respiratory cycle.

9. Residual volume - The volume of gas remaining in the lungs at the end of a maximal expiration.
10. Vital capacity (VC) - The largest volume measured on complete expiration after the deepest inspiration without forced or rapid effort.
11. Forced vital capacity (FVC) - The largest volume measured on complete expiration after the deepest inspiration with a forced and rapid effort.

Basic Assumptions

The following assumptions were made at the onset of this investigation:

1. The subjects performed a true maximal effort in all pressure measurements.
2. The subjects performed a true maximal effort during both exhaustive exercise trials.
3. Changes in Pe_{max} were due to changes in the function of the expiratory muscles.
4. No significant changes in VO_{2max} occurred between trials.

Summary

In endurance athletes, training may induce adaptations in the respiratory musculature. Endurance athletes display greater ventilatory endurance than non-athletes (Martin & Stager, 1981). But it is not known whether the fatiguability of the expiratory muscles is different in trained and untrained people. No definitive answer is available yet, but it is possible that elite athletes have improved their cardiovascular and locomotor abilities so that these may no longer be the limiting factors during exhaustive exercise. Therefore, fatigue their respiratory muscles may come into play. (Dempsey, 1986). On the other hand, the respiratory muscles are also skeletal muscles and adapt as such. Training by running or cycling will not only improve the ability of the limb muscles to extract and use oxygen but also the ability of the respiratory muscles to do the same. This is demonstrated by the biochemical and histochemical changes that occur in the respiratory muscles after endurance training. Although Coast et al., (1990) found a difference in inspiratory muscle fatigue between trained and untrained subjects, the mechanisms behind these changes brought on by training are not clear. This study will investigate the limits of the expiratory muscles in trained cyclists and will a comparison between trained and untrained subjects.

Chapter II

REVIEW OF LITERATURE

The respiratory system is responsible for operating under varying conditions. During peak aerobic exercise, ventilation can increase 20 times resting levels (Whipp & Wasserman, 1991). Because of the demands put on it, the adaptability of the respiratory system is extremely important. This review will examine the respiratory muscles and their performance in exercise, the effects of chronic obstructive pulmonary disease (COPD) on respiratory muscles, and inspiratory muscle and expiratory muscle performance. Specifically, it will examine the research on the effects of loaded breathing and chronic and acute exercise on inspiratory and expiratory muscle performance.

The Respiratory System

The main function of the respiratory apparatus is to move air in and out of the lungs. The muscles of inspiration, the external intercostals, internal obliques, and the diaphragm, contract to elevate the rib cage and lower the diaphragm. The intrathoracic volume increases while the pressure decreases and air rushes into the lungs (Gardner, 1975). The muscles of expiration, the internal intercostals, rectus abdominus, external and internal obliques and transversus abdominus, can contract to squeeze the rib cage inward and force air out (Gardner, 1975), but they generally do not act during passive breathing (Derenne, 1978). Rather, during passive expiration in healthy people, the elastic properties of the lung and the relaxation of the inspiratory muscles are enough to slowly empty the lungs but not all the way to residual volume. The expiratory muscles are called upon for expulsive efforts like coughing (Agostoni,

1964) and during exercise at ventilations above about 40 L/min (Strohl, Mead, Banzett, Loring & Kosch, 1981) which would correspond to moderate activity. Unlike healthy people, COPD patients must use their expiratory muscles even during passive expiration to force air out of their constricted respiratory system.

Respiration during exercise

During exercise the respiratory system must increase oxygen delivery to the blood and also increase elimination of carbon dioxide. The body meets these demands by increasing ventilation, altering tidal volume, increasing breathing frequency, increasing gas transfer and increasing pulmonary blood flow (Stubbing, Pengelly, Morse & Jones., 1980). Breathing dynamics can be examined with flow-volume relationships or loops, which are plots of airflow during the volume changes of the breathing cycle. As exercise intensity increases, flow-volume loops become larger (Beck, Babb, Staats & Hyatt, 1991) indicating an increase in the amount of airflow, possibly due to bronchodialation (Stubbing, et al., 1980), and an increase in tidal volume (Dempsey, 1986). Some researchers have found some individuals in whom the maximum expiratory flow volume (MEFV) curve during exercise exceeds their MEFV curve at rest (Olafsson & Hyatt, 1969). This would indicate an ability to ventilate during exercise that is far beyond what is voluntarily possible. Others, however, find that MEFV curves during exercise are equal to or do not reach the MEFV at rest and postulate that Olafsson & Hyatt's findings are in error because they did not account for changes in lung volumes (Stubbing et al., 1980).

How well does the respiratory system meet the demands exercise puts on it? In normal, healthy adults participating in heavy short term exercise, the pulmonary system can adequatelymeet the added metabolic and homeostatic demands placed on it (Dempsey, 1986). On the other hand, it is widely accepted that the respiratory muscles, like other skeletal muscles have a limited ability to sustain high ventilations (Keens, Krastins, Wannamaker, Levison, Crozier & Bryan, 1977; Tenney & Reese, 1968). In normal healthy individuals, however respiratory muscle fatigue generally is not a problem because their VO_{2max} is dependent on other factors such as maximum stroke volume, cardiac output, skeletal muscle vascularity , or oxidative capacity of skeletal muscle (Dempsey, 1986). It is only in the elite athlete who has trained to improve these cardiovascular factors, that the maximum ventilation during exercise may approach MVV which implies a mechanical ventilatory limit (Dempsey, 1986). In fact, Dempsey hypothesizes that there are two different models of $VO2_{max}$ limitation, one for average untrained or moderately trained people and one for highly trained athletes. He hypothesizes that in the untrained the ability of the cardiovascular system to transport oxygen and the oxidative capacity of the skeletal muscles lags behind the capacity of the pulmonary system for oxygen transport. Upon training, the skeletal muscles and the cardiovascular system adapt and can become highly efficient at delivering and extracting oxygen. Only when a person has trained to the point where the pulmonary system can no longer meet the demands of the limbs and cardiovascular system is the pulmonary system a limit to performance (Dempsey, 1986).

These hypotheses were made in regard to healthy people. In patients with obstructive airway diseases, such as COPD, these ideas do not hold. The muscles of both inspiration and expiration must work harder to force air in and out of a diseased lung and so the muscles require more energy and more oxygen. Eventually, as the disease progresses, the respiratory system, including the respiratory muscles, become the limiting factor in performing everyday activity for the patient. It is unknown whether it is respiratory muscle fatigue that leads to eventual respiratory failure or whether it is some other factor or combination of factors. COPD will be discussed in greater detail later in this review.

Trainability of respiratory muscles

Since the lungs are not normally a limiting factor in exercise for healthy individuals, training the respiratory muscles would not be a consideration for most people. But respiratory muscle training may be of interest to an elite athlete who wants to improve performance. Training of respiratory muscles is also of interest to researchers in clinical settings studying COPD patients. But can the respiratory muscles be trained like other skeletal muscles?

One of the earliest studies on respiratory muscle training examined specifically strength and endurance training in normal subjects (Leith & Bradley, 1976). Strength trainers performed 5 weeks, 5 days/week, 30-45 min/day of repeated static inspiratory and expiratory maneuvers against obstructed airways. Endurance trainers spent the same amount of time performing voluntary hyperpnea to exhaustion in an isocapnic environment, or an environment with a constant concentration of CO_2. Strength trainers increased their pressure maximums by 55% but increased their vital capacity and total lung capacity by only 4%. Endurance trainers increased their maximum voluntary ventilation (MVV) by 14%. It appears as though ventilatory muscle strength or endurance can specifically be increased by training and pressure maximums especially respond to training.

Although Leith & Bradley showed that ventilatory muscle training improves performance during voluntary ventilatory maneuvers, that does not mean that it actually improves respiratory performance in situations with heavy ventilatory loads such as exhaustive exercise or in lung disease (Leith, Philip, Gabel, Feldman & Fencl, 1979). Keens, et al. (1977) feel that because muscle endurance is correlated with resistance to fatigue, improving respiratory muscle endurance may help patients with COPD. Although the research in this area is lacking, Keens, et al. (1977) examined ventilatory muscle endurance training in cystic fibrosis (CF) patients. They compared ventilatory endurance of normal subjects and CF patients by having them perform a isocapnic hyperpnea test lasting 15 minutes and found that the CF patients had 36% higher ventilatory muscle endurance than normals subjects. This may reflect a training effect in the CF patients from chronically breathing against increased respiratory loads caused by their disease. Subjects, both healthy and with CF, then trained with isocapnic hyperpnea 25 min/day, 5 days/week, for 4 weeks. The CF patients improved respiratory endurance 51.6% while the normal subjects improved only 22.1%. It may be that the same hyperpnea load in CF patients provided a greater workload for the respiratory muscles because of the airway obstruction. A greater workload would lead to greater performance gains.

Robinson & Kjeldgaard (1982) showed that ventilatory muscle training can be accomplished by running as well as by specific ventilatory maneuvers. Healthy volunteers participated in a 20 week running program. They found increases in maximum sustainable ventilatory capacity for 15 minutes (MSVC), maximum voluntary ventilation (MVV) and maximum expiratory pressure (Pe_{max}). This demonstrates an increase in both strength and endurance of ventilatory muscles simply by training with running only.

It has also been shown that ventilatory muscle training by running not only improves respiratory maneuver performance but also results in biochemical changes in the muscle fibers. Moore & Gollnick (1982) examined biochemical properties of the muscle fibers of rat diaphragm and intercostal muscles after training by treadmill running. They found increased succinate dehydrogenase (SDH), which is used to estimate oxidative potential of a muscle, in the diaphragm. But they found no change in SDH in the intercostals. These results may mean that the ventilatory muscle endurance training affect seen in the previous studies was not generalized to all respiratory muscles but only to certain muscles such as the diaphragm. Likewise, Uribe, et al. (1992) found that endurance training in rats led to increases in citrate synthase (CS), also a measure of oxidative capacity, in locomotor muscles and the diaphragm but not in the abdominal expiratory muscles. Thus, changes in the respiratory musculature are not uniform but rather occur predominantly in the diaphragm which is an inspiratory muscle.

Obstructive Lung Diseases

COPD patients generally describe the major symptom of their disease as breathlessness. COPD causes reduced ventilatory capacity (Cotes, Reed & Elliott, 1991) and increased work of breathing causing the pulmonary system to limit the ability of COPD patients to perform work (Suzuki, et al., 1991). Several breathing abnormalities have been noted in association with COPD including: abnormal movement of the rib cage and abdomen (Macklem & Roussos, 1977), abnormal diaphragm motion, increased use of scalene and sternomastoid muscles and disuse of the abdominal expiratory muscles (Druz, Danon, Fishman, Goldberg, Moisan & Sharp, 1979). These changes in respiratory function are more closely related to the increase in lung volume, or hyperinflation of the lung, associated with COPD than to the airway obstruction (Druz, et al., 1979; Roussos, Fixley, Gross & Macklem, 1976). The inspiratory muscles are put at a mechanical disadvantage in COPD since they are stretched by the increased lung volume making it more difficult for them to contract (Braun & Rochester, 1977). COPD patients in advanced stages of the disease also suffer from general muscle weakness and debilitation which affects the abilities of the respiratory muscles. It has been shown that fatigue of the respiratory muscles can be experimentally produced in healthy people by having them breath against a respiratory load (Suzuki et al., 1991). Therefore, there is a potential in every COPD patient to experience respiratory muscle fatigue and possibly respiratory failure as a result of breathing against the resistance of the lung obstruction (Roussos, Fixley, Gross & Macklem, 1979).

Respiratory muscle fatigue is thought to be one of the factors that contribute to respiratory failure (Macklem & Roussos, 1977; Suzuki et al., 1991) Some researchers report that patients with COPD show no decreases in expiratory muscle strength (Byrd & Hyatt, 1968) while others report a reduced expiratory and inspiratory muscle strength (Rochester, Braun & Arora, 1979; Braun & Rochester, 1977). In fact, as was stated earlier, Keens et al. (1977) reported increased respiratory muscle endurance in patients with cystic fibrosis which is also an obstructive lung disease. The differences may be due to the type and severity of the disease and also to the patient's age and overall health and nutritional status. In young CF patients the respiratory muscles generally improve their efficiency while in older patients the respiratory muscles become less efficient (Campbell, Hughes, Sahgal, Frederiksen & Shields, 1980).

Patients with COPD can improve their respiratory muscle function and exercise performance. Pardy, Rivington, Despas & Macklem (1981) found that inspiratory muscle training by inspiring against a resistance 30 min/day improved exercise endurance in patients with chronic airflow limitations. Since inspiratory muscle training improved exercise performance in these patients it seems likely that the respiratory muscles were weak. The muscle weakness may have been due to debilitation from a greatly reduced activity level. But, it is not known whether respiratory muscle debilitation leads to eventual respiratory failure or whether some other factor or combination of factors is responsible. As COPD patients age and their disease progresses, breathing becomes more difficult. Braun & Rochester (1979) found a decrease in vital capacity (VC), MVV, Pi_{max}, and Pe_{max} in patients with myopathic processes of the pulmonary system. They in fact developed an index of respiratory muscle strength (RMS) which averages Pi_{max} and Pe_{max} expressed as percentages of predicted normal respiratory pressures. They found that in half of their subjects, RMS was less than 50% of predicted, indicating fairly severe respiratory muscle weakness. The usefulness of the RMS index is unknown, however, because normal respiratory pressures are difficult to determine since even in healthy people respiratory pressures vary considerably (Byrd & Hyatt, 1968).

COPD and other pulmonary diseases may also cause changes in respiratory muscle biochemistry which may indicate a mechanism of respiratory muscle fatigue or failure. Campbell et al. (1980) examined intercostal muscle morphology in patients with obstructive lung diseases. They found muscle fiber atrophy and decreased ATP and phosphocreatine concentration. Fiber atrophy was related to the degree of airway obstruction in the patient. Farkas & Roussos (1984) feel that the current research indicates that the diaphragm adapts to chronic lung hyperinflation associated with COPD first by increasing fiber size to maintain respiratory pressure under adverse conditions. After hyperinflation becomes severe, atrophy of the diaphragm fibers occurs and patients can not maintain respiratory pressures. In their study on emphysematous hamsters, Farkas & Roussos found that aerobic exercise helps prevent some loss of muscle strength and pressure development as the disease progresses.

Exactly how hyperinflation of the lung, respiratory muscle fatigue and muscle atrophy all come together to lead to respiratory failure has not been definitively answered. More research into respiratory function with airflow limitation is needed to determine the dynamics at work in patients with COPD.

Inspiratory Muscles

The inspiratory muscles are extremely important because they are always in use. They can be voluntarily controlled but also must contract involuntarily when needed. They must be able to work continually with little rest and without fatiguing. In a study that compared inspiratory and expiratory muscle endurance to that of the flexor and extensors of the elbow, the inspiratory muscles fatigued less than any of the locomotor muscle groups. After a series of sustained contractions separated by one minute rest intervals, the expiratory muscles and the flexors and extensors of the elbow showed a decline in force. The inspiratory muscles, however, recovered their ability to generate force completely in that one minute rest period (Gandevia, McKenzie & Neering, 1983).

Loaded breathing

Studies examining loaded breathing and its effect on inspiratory muscle performance and fatigue help demonstrate the limits of the respiratory muscles. Roussos et al, (1979) studied the time to produce inspiratory muscle fatigue by having subjects breath against a variety of high inspiratory resistive loads. Subjects generated a mouth pressure (Pm) that was a predetermined fraction of maximum mouth pressure (Pm_{max}). The Pm/Pm_{max} that could be generated indefinitely was found to be 60%, beyond that fatigue occurred. However, Roussos & Macklem (1977), found that the same threshold was only 40% of maximum pressure. Other studies also find various limits (Gross, Grassino, Ross & Macklem, 1979; Bellemare & Grassino, 1982) possibly due to various inspiratory times. But all the studies do demonstrate that the inspiratory muscles can be fatigued if the load is great enough.

Roussos, et al. (1979) also recorded diaphragmatic EMG during loaded breathing and found a shift in the power spectrum of the EMG to the lower frequencies during the fatiguing work and this shift occurred before fatigue was noticeable as a failure to achieve mouth pressure. Detecting the power spectral shift to lower frequencies of the surface EMG is a reliable way to detect fatigue long before a decrease in power output is detected (Mills, 1982). During the fatiguing bouts the diaphragm and the intercostals alternated their involvement in inspiration against the resistance, possibly postponing the onset of fatigue. This may be a protective mechanism invoked by the respiratory system under stressful conditions to preserve respiration.

Chronic increased respiratory load also leads to cellular changes in respiratory muscle fibers. Keens, Chen, Patel, O'Brien, Levison & Ianuzzo, (1978) produced a chronic respiratory load in rats by tracheal banding. Oxidative capacity of the inspiratory muscles, as indicated by SDH activity, increased. The capacity for beta-oxidation of fatty acids also increased. Glycolitic capacity, however, did not change. All of the increases indicate changes generally associated with an improved endurance and aerobic capacity, although endurance abilities were not measured.

Demedts & Anthonisen (1973) examined the effects of inspiratory loaded breathing on exercise performance. Subjects cycled with and without added airway resistance. Two different levels of resistance were used. The lower level doubled the work of breathing and the higher was considerably larger. The lower level of resistance had little effect on exercise performance. The higher load greatly reduced ventilation and limited the maximum work performed. Thus it takes a considerable inspiratory resistive load to cause a noticeable decrease in exercise performance. Although EMGs were not recorded, if they were we may have seen evidence of fatigue as a shift in the power spectrum of the EMG during the exercise with the lower resistance.

Exercise

Since it has been shown that inspiratory muscle fatigue can be induced in healthy people by loaded breathing, it is logical to investigate the possibility of exercise inducing inspiratory muscle fatigue. Bender & Martin (1985) examined maximal ventilations after exhausting exercise in runners and non-runners by performance of isocapnic 60s MVVs before, during and after short-term exercise (3-10 min) and long-term exercise (60 min). The short-term exercise did not change the 60s MVV in either group but non-runners showed decreased MVVs during the final minute of the 60 minute exhausting exercise and 5 and 10 minutes after exercise. The runners had a normal MVV after the long term exercise bout but had a decreased MVV 10 minutes after exercise. These results indicate that impairment of ventilatory capacity occurs only in long-term exhausting exercise and is a factor for untrained people more than trained.

Ventilation is an important measure of respiratory function but respiratory pressures after exercise are also of interest because they are a more direct measure of respiratory muscle function. Younnes & Kivinen (1984) examined inspiratory pressures after a maximal exercise test. Subjects were given an incremental exercise test to exhaustion. After this short period of exercise, Pi_{max} remained at pre-exercise levels. These subjects were all relatively untrained and probably were limited by cardiovascular or muscular factors before any respiratory muscle fatigue could occur. Coast, Clifford, Henrich, Stray-Gundersen & Johnson (1990) compared the effects of a maximal cycling exercise test on Pi_{max} between trained and untrained subjects. Untrained subjects showed decreases in Pi_{max} after exercise while trained subjects did not. Whether this difference occurred because of a protective effect of training or whether the trained subjects had a genetic predisposition to be resistant to inspiratory muscle fatigue in unclear from this study.

Bye, et al. (1984) found evidence of diaphragmatic fatigue following high intensity, short-term exercise in both room air and 40% O2 in normal men. They found a shift in the power spectrum of the diaphragmatic EMG following an exercise bout at 80% of maximum power output in room air They also found a drop in transdiaphragmatic pressure (Pdi) that recovered by 5 minutes post exercise in 40% O2 but did not recover at 5 minutes in room air. Pemax, however, did not change following exercise. It may be that the diaphragm, an inspiratory muscle, was affected by the short term, high intensity bout while the muscles of expiration were not.

Long term exercise such as a marathon is a different kind of stress on the respiratory system than short term exercise. A marathon will deplete energy supplies in most major muscles. It is of interest to investigate the effects of ventilation during a short bout of exercise on respiratory muscle function. Loke, et al. (1982) studied respiratory muscle function after marathon running by using maximal respiratory pressures to assess strength and MVV to assess endurance. They observed decreases in post race Pi_{max}, Pe_{max} and MVV. They concluded that these results indicate development of respiratory muscle fatigue after the marathon.

Inspiratory muscle fatigue and performance

If inspiratory muscle fatigue does occur, it is not of any significance unless it has an effect on a person's ability to perform work. Mador & Acevedo (1991a) examined the effect of respiratory muscle fatigue on exercise performance based on exercise time. They induced respiratory muscle fatigue by having normal subjects breath against an inspiratory threshold while generating 80% of maximum mouth pressure until subjects could not reach the target pressure. Induction of inspiratory muscle fatigue reduced total exercise time and VO2max. Also the subjects' breathing patterns changed to rapid and shallow after fatigue. Another study by Mador & Acevedo (1991b) investigating incremental exercise after respiratory muscle fatigue, also found alterations in breathing pattern and found rapid but not shallow breathing at higher workloads, but found that $VO2_{max}$ was not altered.

Expiratory Muscles

Loaded breathing

The effects of loaded breathing on expiratory muscle performance have not been studied as extensively as the effects on inspiratory muscle performance. Suzuki, et al. (1991) studied expiratory muscle fatigue during expiratory resistive loading by having subjects breath against varying expiratory resistances at their own breathing frequency and tidal volume until exhaustion or for 60 minutes. They then assessed Pi_{max} and Pe_{max}. At the highest resistance both Pi_{max} and Pe_{max} were decreased and remained decreased for at least 60 minutes after resistive breathing. They also recorded EMG of the rectus abdominis and found that the high-to-low frequency power ratio decreased progressively and transdiaphragmatic pressure increased during loaded breathing. The results suggest that expiratory resistive loading causes expiratory and inspiratory muscle fatigue and specifically fatigue of the rectus abdominis muscle and a decrease in the diaphragm's ability to generate pressure. The fatigue of the expiratory muscles is probably due to the increased work load, but the inspiratory muscle fatigue is more difficult to explain since expiratory loads were used. It may be due to increased work due to shortened inspiratory time or it may be due to generalized energy depletion because of exhaustion.

Exercise

Inspiratory muscles have been shown to fatigue after exhausting exercise, therefore, the possibility exists that expiratory muscles will also fatigue with either long or short term exercise. Maron, Hamilton & Maksud, (1979) investigated the effects of marathon running on respiratory measures. Their results do not support an expiratory muscle fatigue mechanism. They found a reduction in forced vital capacity (FVC) after a marathon race, but since there was no change in forced expiratory flow between 200 and 1200 something ($FEF_{200-1200}$), which would indicate some expiratory muscle fatigue, they theorize that the reduction in FVC is due to small airway closure at increased lung volumes and not to muscle fatigue. Mahler & Loke (1981), however, have a different view. They looked at mean expiratory flow volume (MEFV) curves before and after a 100 km road race. They saw decreases in FVC, FEV_1 and peak expiratory flow rate after the race with all factors improving at 2.5 hours post-race. They also hypothesized that the reduced flow rates were due to airway obstruction. But they felt that the decrease in FVC may be in part due to respiratory muscle fatigue, because respiratory muscle fatigue can cause the decreases seen in FVC and FEV_1. The FEV_1/FVC ratio remained constant after the race negating the possibility of large airway closure. Small airway closure, however, can not be eliminated. No direct measures of respiratory muscle function, such as respiratory pressures, were made.

Neither Mahler & Loke or Maron, et al. actually measured expiratory pressures after exhaustive exercise so the direct effect of long duration exercise on the expiratory muscles can not be known . Loke, et al. (1982) measured expiratory pressures and MVV after a marathon. They found decreases in both maximum expiratory pressure and MVV post-race. Although only 4 subjects were used, these results suggest fatigue of the expiratory muscles occurs following exhaustive exercise. No other recovery data were available.

Bye, et al. (1984) also examined Pe_{max} in addition to transdiaphragmatic pressure (Pdi) and diaphragmatic EMG before and after exercise They, however, used a short term, high intensity bout of exercise. Untrained subjects cycled at 80% of their maximal power output. They examined these factors in air and in 40% O_2. They found that Pdi decreased post-exercise in air and O_2, but recovered at 2-5 minutes post exercise completely in O_2 but not in air. Diaphragmatic EMG predicted diaphragm fatigue, using a 20% power ratio decline as an index, in 5 out of 7 subjects. They found no changes in either air or O_2 in Pe_{max} post exercise. These results indicate that diaphragm fatigue is occurring but no detectable expiratory muscle fatigue accompanies it.

Cerny, Calhoun & Reinstein, (1991) tested the effects of abdominal muscle fatigue, brought on by sit ups to exhaustion, on Pe_{max}. They found no change in Pe_{max} after abdominal muscle fatigue. No EMGs were recorded, however, to verify fatigue and it is possible that some of the expiratory the abdominal muscles were not exhausted during the sit ups and thus subjects were able to maintain Pe_{max}.

Trained vs. untrained

It has been seen that in endurance athletes, training may induce adaptations in the inspiratory musculature. Endurance skiiers are able to maintain their maximum inspiratory pressure after exercise whereas non-athletes are not (Coast, et al., 1990). Endurance athletes also display greater ventilatory endurance than non-athletes (Martin & Stager, 1981). But is the fatiguability of the expiratory muscles different in trained and untrained people? No definitive answer is available yet, but two theories can be discussed. Elite athletes have improved their cardiovascular and locomotor abilities to their peak performance and thus during exhaustive exercise of long duration they may fatigue their respiratory muscles (Dempsey, 1986). On the other hand, the respiratory muscles are also skeletal muscles and adapt as such. Training by running or cycling will not only improve the ability of the limb muscles to extract and use oxygen but also the ability of the respiratory muscles to do the same. Thus the respiratory muscles are better trained in endurance trained people than in untrained people (Robinson & Kjeldgaard, 1982).

Summary

The respiratory muscles have been shown to adapt to specific muscle training and also to endurance training. Both the inspiratory and expiratory muscles can be fatigued with loaded breathing and long duration exercise. Respiratory muscle fatigue may also play a role in respiratory failure in patients in the later stages of obstructive lung diseases. The effect of short term exercise on respiratory muscle fatigue is not clear. Various researchers have found different results in both athletes and non-athletes after short term exercise. This is an area in which more research needs to be done to determine the limits of the respiratory muscles and how exercise affects them.

Chapter III

JOURNAL MANUSCRIPT

Evaluation of Maximum Expiratory Pressures Following a High Intensity Exercise Bout in Highly Trained Individuals

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Evaluation of Maximum Expiratory Pressures Following a High Intensity Exercise Bout in Highly Trained Individuals

ABSTRACT

Ten well trained cyclists were studied and compared with 12 untrained subjects from a previous study to determine the effects of a high intensity, constant workload bout of cycling on maximum expiratory pressure (Pe_{max}). Subjects completed a graded exercise test on a Monark cycle ergometer while expired gases were collected to determine maximal oxygen consumption (VO_{2max}). Subjects then returned on a second day when measurements of each subject's Pe_{max} were made prior to riding at the workload corresponding to 90% of their VO_{2max} until exhaustion. Measurements of expiratory pressure (Pe) were then made immediately post exercise (Pe_{IPE}), one minute post exercise (Pe_{1MIN}), three minutes post exercise (Pe_{3MIN}), and five minutes post exercise (Pe_{5MIN}). Trained cyclists had a significantly higher Pe_{max} (x = 116.43 ± 7.76 mmHg) than did untrained subjects (x = 65.75 ± 7.09 mmHg). Also trained cyclists generated a higher absolute Pe throughout recovery than did the untrained subjects. Although expiratory pressure decreased after exercise in both groups, the relative change in Pe over the recovery period, expressed as a percentage of Pe_{max}, was not different between trained and untrained. Pe_{IPE} was decreased to 81.87% ± 3.12 of Pe_{max} in trained subjects and 82.35% ± 2.85 in untrained subjects ($p<.05$), recovering somewhat at 1 minute to 89.19% ± 3.59 of Pe_{max} in trained and to 87.74% ± 3.27 in untrained($p<.05$) but did not recover to resting levels in either group. Pe_{3MIN} and Pe_{5MIN} remained at the same level as Pe_{1MIN} in both groups. Therefore, a high intensity, short term exercise bout caused expiratory pressure to be decreased in both trained and untrained subjects.

Introduction

Most of the research in the area of respiratory muscle fatigue has concentrated on inspiratory muscle fatigue. During exercise, however, the expiratory muscles also play an important part by providing the quick, forced exhalation needed to increase ventilation rates to levels needed during aerobic exercise. Expiratory muscle fatigue, therefore, is of extreme interest in the study of the limits of the respiratory system in exercise.

What little research has been done in the area of expiratory muscle fatigue, however, has concentrated on the effects of loaded breathing or long distance running on expiratory muscle performance. Suzuki, Suzuki & Okubo (1991) reported a decrease in maximum expiratory pressure (Pe_{max}) for up to 60 minutes following expiratory resistive loaded breathing. But, expiratory resistance does not come into play normally during exercise. Rather, during exercise, the stressor on the respiratory muscles is the high ventilation rates. Loke, Mahler & Virgulto, (1984) examined the effects of long duration exercise on maximum expiratory pressures. They found a decrease in expiratory pressures after a marathon race. This indicates that a long term exercise bout may cause expiratory muscle fatigue in highly trained runners, possibly due to the sustained high ventilation rates. Therefore, the purpose of this study was to determine if expiratory muscle fatigue will be detected as a reduction in Pe_{max} in trained cyclists or untrained subjects after a short term, high intensity endurance ride at 90% of VO_{2max}.

Methods

Ten trained cyclists, who were actively cycling 100 miles per week, and 12 untrained subjects, age 20-32, acted as subjects. Subjects were all nonsmokers with no history of chronic pulmonary disease and no recent upper respiratory infections. Subjects were screened by completing a questionnaire. $FEV_{1.0}$ values were measured both before and after exercise to eliminate any subjects with present pulmonary dysfunction. Trained subjects performed a graded exercise test on a cycle ergometer starting at 50 Watts and increasing 50 Watts every 2 minutes until exhaustion. Untrained subjects performed a similar test but were increased only 25 Watts per stage. Expired gases were collected via open circuit spirometry using a Parkinson-Cowen dry gas meter to measure ventilation and a Metex S-3A oxygen analyzer and a Metex CD-3A carbon dioxide analyzer to compute VO_2 and VCO_2 in order to determine VO_{2max}. Subjects then returned on a second day for a high-intensity cycling bout at a workload corresponding to 90% of VO_{2max}. Each test required subjects to exercise to voluntary exhaustion. Measurements of Pe_{max} were taken before the 90% exercise bout, immediately post exercise and at 1 minute, 3 minutes and 5 minutes into recovery.

After a 3 minute warm up, subjects cycled at a workload corresponding to 90% of their VO_{2max} until they were unable to continue despite verbal encouragement. During this exercise trial, heart rate and blood pressure were determined. Pe_{max}, was measured before and after the exercise trial through the use of a resistance tube attached to a mouthpiece which leads into a pressure transducer (Omega Engineering, 0-250 mmHg). A small leak was present in the tube in order to prevent glottic closure. Pressure signals were amplified (Grass P511 low-level DC, 0-30 Hz) and displayed on a strip-chart recorder (Kipp & Zonen). Prior to each test, the transducer was calibratedby applying known pressures with a sphygmomanometer and monitoring the pen displacement on the strip chart recorder. Subjects, while wearing a noseclip, inhaled to total lung capacity. The airway on the apparatus was closed and each subject exhaled maximally against the resistance tube. A minimum of three resting trials was obtained before the 90% bout. More than three trials were performed if significant variation exist between the second and third trials. Measurements of Pe were also taken after the exercise bout, including within twenty seconds of test termination (Pe_{IPE}), and 1 minute (Pe_{1MIN}), 3 minutes (Pe_{3MIN}) and 5 minutes (Pe_{5MIN}) after test termination. Subjects were verbally encouraged to give a maximal effort during each trial.

Results

Results of the incremental exercise tests revealed that trained subjects had a higher VO_{2max} (x = 61.71 ± 1.95 ml • kg-1 • min-1) than untrained subjects (x = 40.00 ± 2.32 ml • kg-1 • min-1) (P<.05). Trained subjects also had a higher Pe_{max} (x = 116.43 ± 7.76 mmHg) than untrained subjects (x = 65.75 ± 7.09 mmHg) (P<.05).

Results of a repeated measures ANOVA show that both trained and untrained subjects have a significant decrease in expiratory pressure immediately following the exercise bout at 90% of VO_{2max} recovering somewhat at 1 minute but remaining decreased throughout 5 minutes of recovery (p < .05) (See Fig. 1). In trained subjects, Pe_{IPE} was decreased from 116.43 ± 7.76 to 94.42 ± 6.01, Pe_{1MIN} recovered somewhat to 102.92 ± 6.60 and remained at the same levels with Pe_{3MIN} at 105.74 ± 6.26 and Pe_{5MIN} at 105.21 ± 7.36. In untrained subjects there was a similar pattern with PeIPE decreased from 65.75 ± 7.09 to 54.28 ± 5.48, Pe_{1MIN} recovered somewhat to 57.84 ± 6.02 and Pe_{3MIN} remained at 58.29 ± 5.71 and Pe_{5MIN} at 58.16 ± 6.72.

The relative decrease in expiratory pressure following exercise, expressed as percent of Pe_{max}, was not different between trained and untrained subjects (p<.05) Immediately after exercise, Pe_{IPE} in trained subjects decreased to 81.87% ± 3.12 of Pe_{max} while Pe_{IPE} in untrained subjects decreased to 82.35% ± 2.85 of Pe_{max}. Pe_{1MIN} in trained subjects was 89.19 % ± 3.59 of Pe_{max} and Pe_{1MIN} in untrained subjects was 87.74% ± 3.27 of Pe_{max}. Pe_{3MIN} in trained subjects was 92.20% ± 3.11 of Pe_{max} and Pe_{3MIN} in untrained subjects was 87.55% ± 2.84 of Pe_{max}. Finally, Pe_{5MIN} in trained subjects was 90.97% ± 3.12 of Pe_{max} and Pe_{5MIN} in untrained subjects was 86.52% ± 2.85 of Pe_{max} (Fig. 2).

VO_{2max} was not significantly correlated to percent decline in Pe_{max} (see Fig. 3). Correlation coefficients ranged from -0.03 for percent decline at IPE to 0.31 for percent decline at 5 minutes. Maximum ventilations (Ve_{max}) were also not correlated to percent decline in Pe_{max} (see Fig. 4). Correlation coefficients ranged from 0.05 for percent decline at IPE to 0.43 for percent decline at 5 minutes.

Discussion

It can not be determined from this study whether the higher Pe_{max} measurements seen in trained subjects is a direct result of training or a function of genetics or some other factors. In addition to the higher expiratory pressures shown in this study, athletes have been found to possess greater ventilatory endurance as measured by a maximum voluntary ventilation (MVV) test to exhaustion (Martin & Stager, 1981). To determine if training is the cause of the improved respiratory function in athletes a study examining subjects before and after a training program must be done. Robinson & Kjeldgaard (1982) showed an improvement in respiratory measures after a 20 week aerobic training program. Subjects improved Pe_{max} by 14.4%, MVV by 13.6% and maximum sustainable ventilatory capacity for 15 minutes (MSVC) by 15.8% after 20 weeks of training.

If respiratory function is improved by training, the mechanism behind the improvement is unknown. A muscle's power and endurance are dependent on both muscle mass and oxidative abilities (Keens & Ianuzzo, 1979). Increasing either of these will improve muscle strength and/or endurance. Powers, Criswell, Lieu & Silverman (1992) found the oxidative capacity of the crural region of the rat diaphragm, as indicated by succinate dehydrogenase (SDH) activity, and capillary density, increased after 10 weeks of training. Moore & Gollnick (1982) also found SDH activity in the rat diaphragm increased after 12 weeks of endurance training but they found no changes in the SDH activity of the intercostals. Metzger & Fitts (1986) found no change in force output, ATP or creatine phosphate (CP) concentration of the diaphragm after only 6 weeks of training in rats.

It is possible that improved SDH activity in the diaphragm accounts for some training adaptations of the respiratory system, but this does not account for the difference in expiratory force between trained and untrained subjects. Uribe, Stump, Tipton & Fregosi (1992) measured citrate synthase (CS) activity to assess oxidative capacity in the abdominal expiratory muscles of rats after 9 weeks of training. They found training increase CS activity only in the rectus abdominis but not in the transversus abdominis, external obliques, or the internal obliques. They felt that the transversus abdominis is used for postural support and locomotion in the rat and the other expiratory abdominal muscles did not contribute to the increased ventilatory load associated with exercise training. Therefore, these results in rats may not hold for humans. More study on the adaptation of the human expiratory muscles to endurance training needs to be done to determine the effects of training and the mechanism that leads to those effects.

In the present study, both groups experienced a decrease in Pe_{max} lasting for at least 5 minutes into recovery from an exercise bout a 90% of VO_{2max} (See Fig. 1). In fact both groups experienced the same relative amount of decrease in Pe_{max} (See Fig.

2). This suggests that although trained subjects show a greater expiratory strength, they do not have the benefit of resistance to expiratory muscle fatigue.

Respiratory muscle fatigue after exercise has been shown before, but most of the research previously focused on inspiratory muscle fatigue and long duration exercise. Several investigators have studied respiratory function following a marathon. Mahler & Loke (1981) studied mean expiratory flow volume (MEFV) curves before and after a 100 km road race. They saw decreases in FVC, FEV_1 and peak expiratory flow rate after the race with all factors improving at 2.5 hours post-race. They hypothesized that the reduced flow rates were due to airway obstruction. But they felt that the decrease in FVC may be in part due to respiratory muscle fatigue, because respiratory muscle fatigue can cause the decreases seen in FVC and FEV_1. The FEV_1/FVC ratio remained constant after the race negating the possibility of large airway closure. Small airway closure, however, can not be eliminated. No direct measures of respiratory muscle function, such as respiratory pressures, were made. Loke, Mahler & Virgulto, 1982 measured respiratory pressures 21 - 60 minutes following a marathon. They found Pe_{max}, Pi_{max} and MVV decreased following the long duration, exhaustive run.

Short term exercise, such as an incremental exercise test is different from a marathon in that glycogen depletion will not occur. Some studies show no evidence of respiratory muscle fatigue following short term exercise. Younes & Kivinen (1984) found that Pi_{max} remained unchanged after an incremental maximal exercise test to exhaustion in untrained subjects. Coast, Clifford, Henrich, Stray-Gundersen & Johnson (1990), however, examined Pi_{max} in cross country skiers and untrained subjects after an incremental exercise test and found decreased inspiratory pressures in untrained subjects but no change in skiers.

Bye, et al. (1984) found evidence of diaphragmatic fatigue following high intensity, short-term exercise in both room air and 40% O_2 in normal men. They found a shift in the power spectrum of the diaphragmatic EMG following an exercise bout at 80% of maximum power output in room air They also found a drop in transdiaphragmatic pressure (Pdi) that recovered by 5 minutes post exercise in 40% O_2 but did not recover at 5 minutes in room air. Pemax, however, did not change following exercise. It may be that the diaphragm, an inspiratory muscle, was affected by the short term, high intensity bout while the muscles of expiration were not.

The current study is the only study so far to find and decrease in Pe_{max} following a short term, high intensity exercise bout. It is also the only study to find any evidence of respiratory muscle fatigue in well trained subjects. Exactly why Bye et al., Younes & Kivinen and Coast et al. did not find any evidence of fatigue after short term exercise whereas the current study found a reduction in Pe_{max} after an endurance ride at 90% of VO_{2max} is unclear. It is probable that Younes & Kivinen did not give their subjects a high enough workload to elicit fatigue. However, Coast, et al. had their untrained subjects complete a similar incremental exercise test and they found a decrease in Pi_{max}.

Bye et al. had their subjects complete an endurance cycling test similar to that used in the present study, the difference was that their subjects rode at 80% of VO2max The subjects in the present study rode until exhaustion at 90% of VO_{2max}. It may be that there is a threshold somewhere between 80% and 90% of VO_{2max} at which expiratory muscle fatigue can be elicited. Unfortunately, neither study measured ventilations during the exercise bout, if they had been measured, a difference may have been noted that could account for the different results. Theoretically, higher sustained ventilations should be more likely to induce fatigue.

In this study, neither VO_{2max} or Ve_{max} were correlated significantly to percent decline in Pe over recovery (See Figs. 3 & 4). This is to be expected since VO_{2max} and Ve_{max} were different between trained and untrained but percent declines in Pe were not different between groups. Since VO_{2max} was not correlated to percent decline in Pe it seems likely that having a higher VO_{2max} does not provide any protection against respiratory muscle fatigue during short term, high intensity exercise bouts. The Ve_{max} in this study refers to the maximum ventilations during the VO_{2max} test and not the exercise bout at 90% of VO_{2max}, therefore, it is not surprising that there is not correlation. Perhaps if ventilations were measured during the 90% exercise bout there would have been a more significant correlation. Although no connection was seen in this study, it is logical that the greater a person's ventilations during an exercise bout, the more they use their expiratory muscles and the more likely it will be that their expiratory muscles will fatigue.

From this study, it is concluded that an exhaustive bout of exercise at 90% of VO_{2max} can cause a reduction in Pe_{max} in both trained and untrained subjects. The relative decrease in Pe is not different between trained and untrained although the absolute Pe values are higher in trained subjects. Future studies examining the changes in muscle fiber morphology and biochemistry before and after training are needed to determine the mechanism behind any training adaptations. More study is also needed to determine if a threshold workload exists beyond which expiratory muscle fatigue occurs.

FIGURE LEGEND

Figure 1. Plot of expiratory pressures at rest and over recovery time for trained and untrained.

● = trained

o = untrained

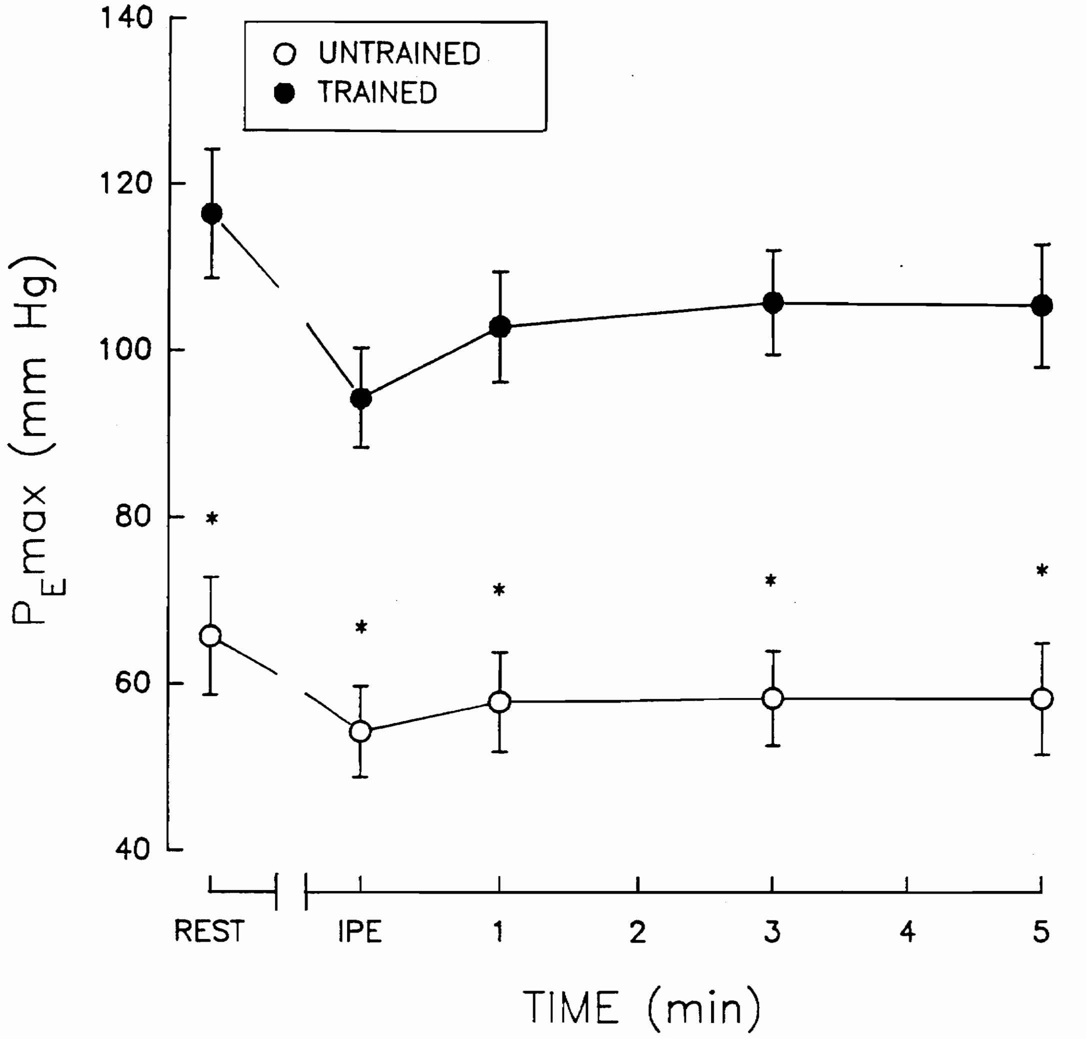
Figure 2. Plot of relative change in expiratory pressure over recovery time, expressed as Pe/Pemax, for trained and untrained.

● = trained

o = untrained

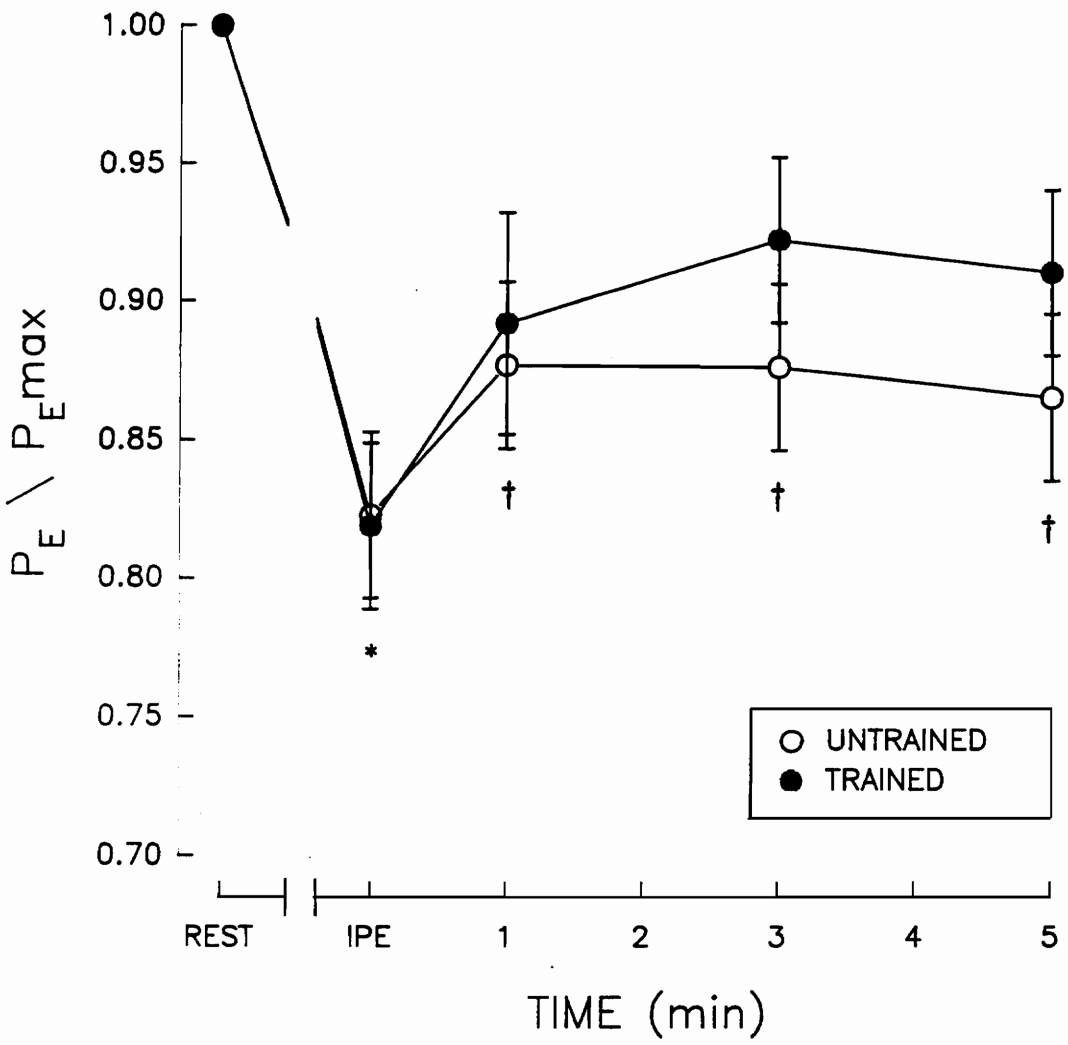
Figure 3. Plot of relationship between VO2max and average change in expiratory pressure at IPE.

Figure 4. Plot of relationship between Vemax during VO2max test and average change in expiratory pressure at IPE.



* difference between groups

Figure 1. Plot of expiratory pressures at rest and over recovery time for trained and untrained.



* different from 1.0
† different from 1.0 and from IPE

Figure 2. Plot of relative change in expiratory pressure over recovery time, expressed as Pe / Pemax, for trained and untrained.

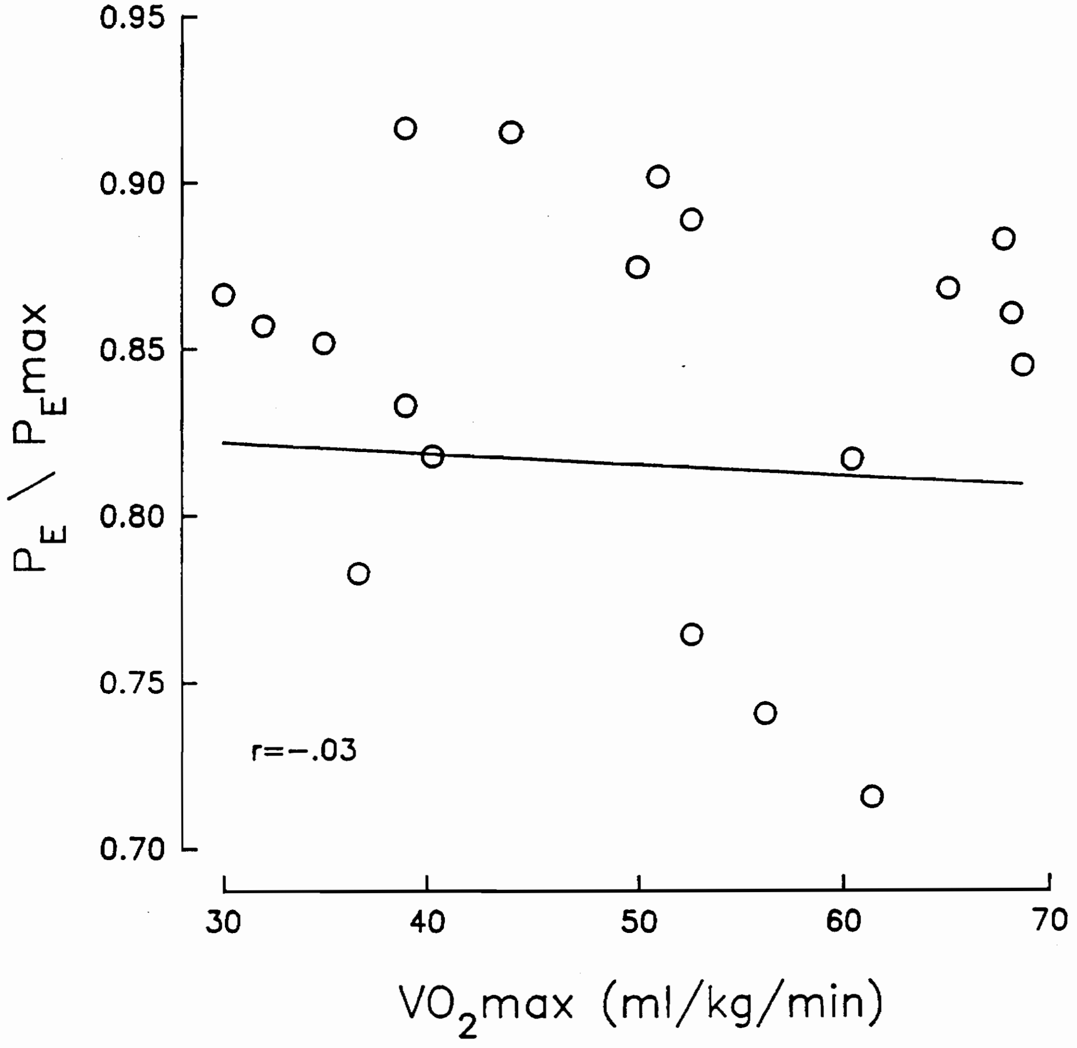


Figure 3. Plot of relationship between VO2max and change in expiratory pressure at IPE.

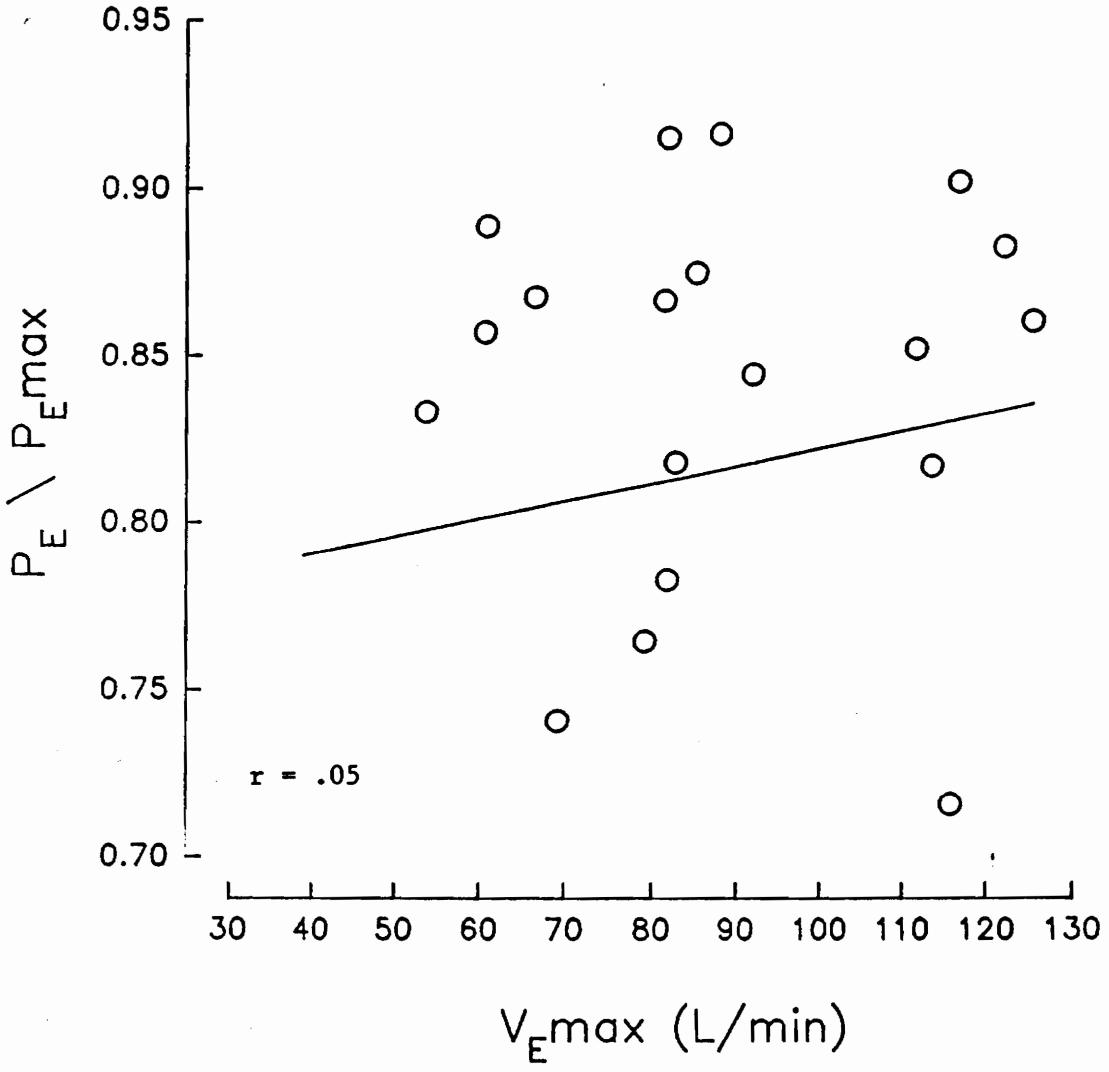


Figure 4. Plot of relationship between Vemax during VO2max test and change in expiratory pressure at IPE.

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Chapter IV

SUMMARY

Aerobic endurance training provides many benefits including improved VO_{2max}, cardiac output, muscle vascularity and oxygen extraction. Whether or not endurance training provides any benefit to the muscles of respiration, such as improved strength or resistance to fatigue, is not clear. Also, if it is possible to fatigue the respiratory muscles of a well trained person with exercise, will only a long term bout of exercise such as a marathon cause fatigue or will a short term, high intensity bout also cause fatigue?

Most of the research in the area of respiratory muscle fatigue has concentrated on inspiratory muscle fatigue. During exercise, however, the expiratory muscles also play an important part by providing the quick, forced exhalation needed to increase ventilation rates to levels needed during aerobic exercise. Expiratory muscle fatigue, therefore, is of extreme interest in the study of the limits of the respiratory system in exercise.

What little research has been done in the area of expiratory muscle fatigue, however, has concentrated on the effects of loaded breathing or long distance running on expiratory muscle performance. Suzuki, Suzuki & Okubo (1991) reported a decrease in maximum expiratory pressure (Pe_{max}) for up to 60 minutes following expiratory resistive loaded breathing. But, expiratory resistance does not come into play normally during exercise. Rather, during exercise, the stressor on the respiratory muscles is the high ventilation rates. Loke, Mahler & Virgulto, (1984) examined the effects of long duration exercise on maximum expiratory pressures. They found a decrease in expiratory pressures after a marathon race. This indicates that a long term exercise bout may cause expiratory muscle fatigue in highly trained runners, possibly due to the sustained high ventilation rates.

Since it is unclear whether expiratory muscle fatigue will occur in trained or untrained subjects after a high intensity short term bout of exercise, this study was designed to investigate the effects of an endurance ride on a cycle ergometer at 90% of VO_{2max} on expiratory pressures in well trained cyclists as well as to compare these results to the results obtained in untrained subjects following the same protocol.

7 male and 3 female trained cyclists, age 20-33, who were actively cycling 100 miles per week acted as subjects. Subjects were also nonsmokers with no history of chronic pulmonary disease and no recent upper respiratory infections. Subjects were screened by completing a questionnaire (see Appendix B). $FEV_{1.0}$ values were measured both before and after exercise to eliminate any subjects with present pulmonary dysfunction. Subjects performed a graded exercise test on a Monark cycle ergometer starting at 50 Watts and increasing 50 Watts every 2 minutes until exhaustion. Expired gases were collected in order to determine VO_{2max}. They then returned on a second day for a high-intensity cycling bout at a workload corresponding to 90% of VO_{2max}. Each test required subjects to exercise to voluntary exhaustion. Measurements of Pe_{max} were taken before the 90% exercise bout, immediately post-exercise and 1 minute, 3 minutes and 5 minutes into recovery.

After a 3 minute warm up at 50 watts, they cycled at a workload corresponding to 90% of their VO_{2max} until they were unable to continue despite verbal encouragement. During this exercise trial, heart rate and blood pressure were determined. Pe_{max} measurements were performed as described previously.

Maximal expiratory pressure, Pe_{max}, was measured through the use of a resistance tube attached to a mouthpiece which leads into a pressure transducer

(Omega Engineering, 0-250 mmHg). A small leak was present in the tube in order to prevent glottic closure. Pressure signals were amplified (Grass P511 low-level DC, 0-30 Hz) and displayed on a strip-chart recorder. Prior to each test, the transducer was calibrated by applying known pressures with a sphyngomanometer and monitoring the pen displacement on the strip chart recorder. Subjects, while wearing a noseclip, inhaled to total lung capacity. The airway on the apparatus was closed and each subject exhaled maximally against the resistance tube. A minimum of three resting trials was obtained before the 90% bout. More than three trials were performed if significant variation exist between the second and third trials. Measurements were also taken after the exercise bout, including within twenty seconds of test termination, and 1, 3 and 5 minutes after test termination. Subjects were verbally encouraged to give a maximal effort during each trial.

Results of the incremental exercise tests revealed that trained subjects had a higher VO_{2max} (x = 61.71 ± 1.95 ml • kg-1 • min-1) than untrained subjects (x = 40.00 ± 2.32 ml • kg-1 • min-1) ($p < .05$). Trained subjects also had a higher Pe_{max} (x = 116.43 ± 7.76 mmHg) than untrained subjects (x = 65.75 ± 7.09 mmHg) ($p < .05$).

Results of a repeated measures ANOVA show that both trained and untrained subjects have a significant decrease in expiratory pressure immediately following the exercise bout at 90% of VO_{2max} recovering somewhat at 1 minute but remaining decreased throughout 5 minutes of recovery ($p < .05$) (See Fig. 1). In trained subjects, Pe_{IPE} was decreased from 116.43 ± 7.76 to 94.42 ± 6.01, Pe_{1MIN} recovered somewhat to 102.92 ± 6.60 and remained at the same levels with Pe_{3MIN} at 105.74 ± 6.26 and Pe_{5MIN} at 105.21 ± 7.36. In untrained subjects there was a similar pattern with Pe_{IPE} decreased from 65.75 ± 7.09 to 54.28 ± 5.48, Pe_{1MIN} recovered somewhat to 57.84 ± 6.02 and Pe_{3MIN} remained at 58.29 ± 5.71 and Pe_{5MIN} at 58.16 ± 6.72.

The relative decrease in expiratory pressure following exercise, expressed as percent of Pe_{max}, was not different between trained and untrained subjects ($p<.05$) Immediately after exercise, Pe_{IPE} in trained subjects decreased to 81.87% ± 3.12 of Pe_{max} while Pe_{IPE} in untrained subjects decreased to 82.35% ± 2.85 of Pe_{max}. Pe_{1MIN} in trained subjects was 89.19 % ± 3.59 of Pe_{max} and Pe_{1MIN} in untrained subjects was 87.74% ± 3.27 of Pe_{max}. Pe_{3MIN} in trained subjects was 92.20% ± 3.11 of Pe_{max} and Pe_{3MIN} in untrained subjects was 87.55% ± 2.84 of Pe_{max}. Finally, Pe_{5MIN} in trained subjects was 90.97% ± 3.12 of Pe_{max} and Pe_{5MIN} in untrained subjects was 86.52% ± 2.85 of Pe_{max} (see Fig. 2).

VO_{2max} was not significantly correlated to percent decline in Pe_{max} (see Fig. 3). Correlation coefficients ranged from -0.03 for percent decline at IPE to 0.31 for percent decline at 5 minutes. Maximum ventilations (Ve_{max}) were also not correlated to percent decline in Pe_{max} (see Fig. 4). Correlation coefficients ranged from 0.05 for percent decline at IPE to 0.43 for percent decline at 5 minutes.

Research Implications

It can not be determined from this study whether the higher Pe_{max} measurements seen in trained subjects is a direct result of training or a function of genetics or some other factors. In addition to the higher expiratory pressures shown in this study, athletes have been found to possess greater ventilatory endurance as measured by a maximum voluntary ventilation (MVV) test to exhaustion (Martin & Stager, 1981). To determine if training is the cause of the improved respiratory function in athletes a study examining subjects before and after a training program must be done. Robinson & Kjeldgaard (1982) showed an improvement in respiratory measures after a 20 week aerobic training program. Subjects improved Pe_{max} by 14.4%, MVV by 13.6% and maximum sustainable ventilatory capacity for 15 minutes (MSVC) by 15.8% after endurance training. Thus, it appears as though training may be at least in part responsible for improved respiratory function.

If respiratory function is improved by training, the mechanism behind the improvement is unknown. A muscle's power and endurance are dependent on both muscle mass and oxidative abilities (Keens & Ianuzzo, 1979). Increasing either of these will improve muscle strength and/or endurance. Powers, Criswell, Lieu & Silverman (1992) found the oxidative capacity of the crural region of the rat diaphragm, as indicated by succinate dehydrogenase (SDH) activity, and capillary density, increased after 10 weeks of training. Moore & Gollnick (1982) also found SDH activity in the rat diaphragm increased after 12 weeks of endurance training but they found no changes in the SDH activity of the intercostals. Metzger & Fitts (1986) found no change in force output, ATP or creatine phosphate (CP) concentration of the diaphragm after only 6 weeks of training in rats.

It is possible that improved SDH activity in the diaphragm accounts for some training adaptations of the respiratory system, but this does not account for the difference in expiratory force between trained and untrained subjects. Uribe, Stump, Tipton & Fregosi (1992) measured citrate synthase (CS) activity to assess oxidative capacity in the abdominal expiratory muscles of rats after 9 weeks of training. They found training increase CS activity only in the rectus abdominis but not in the transversus abdominis, external obliques, or the internal obliques. They felt that the transversus abdominis is used for postural support and locomotion in the rat and the other expiratory abdominal muscles did not contribute to the increased ventilatory load associated with exercise training. Therefore, these results in rats may not hold for humans. More study on the adaptation of the human expiratory muscles to endurance training needs to be done to determine the effects of training and the mechanism that leads to those effects.

In the present study, both groups experienced a decrease in Pe_{max} lasting for at least 5 minutes into recovery from an exercise bout a 90% of VO_{2max} (See Fig. 1). In fact both groups experienced the same relative amount of decrease in Pe_{max} (See Fig. 2). This suggests that although trained subjects show a greater expiratory strength, they do not have the benefit of resistance to expiratory muscle fatigue.

Respiratory muscle fatigue after exercise has been shown before, but most of the research previously focused on inspiratory muscle fatigue and long duration exercise. Several investigators have studied respiratory function following a marathon. Mahler & Loke (1981)studied mean expiratory flow volume (MEFV) curves before and after a 100 km road race. They saw decreases in FVC, FEV_1 and peak expiratory flow rate after the race with all factors improving at 2.5 hours post-race. They hypothesized that the reduced flow rates were due to airway obstruction. But they felt that the decrease in FVC may be in part due to respiratory muscle fatigue, because respiratory muscle fatigue can cause the decreases seen in FVC and FEV_1. The FEV1/FVC ratio remained constant after the race negating the possibility of large airway closure. Small airway closure, however, can not be eliminated. No direct measures of respiratory muscle function, such as respiratory pressures, were made. Loke, Mahler & Virgulto, 1982 measured respiratory pressures 21 - 60 minutes following a marathon. They found Pe_{max}, Pi_{max} and MVV decreased following the long duration, exhaustive run.

Short term exercise, such as an incremental exercise test is different from a marathon in that glycogen depletion will not occur. Some studies show no evidence of respiratory muscle fatigue following short term exercise. Younes & Kivinen (1984) found that Pi_{max} remained unchanged after an incremental maximal exercise test to exhaustion in untrained subjects. Coast, Clifford, Henrich, Stray-Gundersen & Johnson (1990), however, examined Pi_{max} in cross country skiers and untrained subjects after an incremental exercise test and found decreased inspiratory pressures in untrained subjects but no change in skiers.

Bye, et al. (1984) found evidence of diaphragmatic fatigue following high intensity, short-term exercise in both room air and 40% O_2 in normal men. They found a shift in the power spectrum of the diaphragmatic EMG following an exercise bout at 80% of maximum power output in room air They also found a drop in transdiaphragmatic pressure (Pdi) that recovered by 5 minutes post exercise in 40% O2 but did not recover at 5 minutes in room air. Pe_{max}, however, did not change following exercise. It may be that the diaphragm, an inspiratory muscle, was affected by the short term, high intensity bout while the muscles of expiration were not.

The current study is the only study so far to find and decrease in Pe_{max} following a short term, high intensity exercise bout. It is also the only study to find any evidence of respiratory muscle fatigue in well trained subjects. Exactly why Bye et al., Younes & Kivinen and Coast et al. did not find any evidence of fatigue after short term exercise whereas the current study found a reduction in Pe_{max} after an endurance ride at 90% of VO_{2max} is unclear. It is probable that Younes & Kivinen did not give their subjects a high enough workload to elicit fatigue. However, Coast, et al. had their untrained subjects complete a similar incremental exercise test and they found a decrease in Pi_{max}.

Bye et al. had their subjects complete an endurance cycling test similar to that used in the present study, the difference was that their subjects rode at 80% of VO_{2max}. The subjects in the present study rode until exhaustion at 90% of VO_{2max}. It may be that there is a threshold somewhere between 80% and 90% of VO_{2max} at which expiratory muscle fatigue can be elicited. Unfortunately, neither study measured ventilations during the endurance exercise bout, if they had been measured, a difference may have been noted that could account for the different results. Theoretically, higher sustained ventilations should be more likely to induce fatigue.

Although it can be concluded that expiratory muscle fatigue occurred in both trained and untrained subjects following high intensity exercise, the mechanism of the fatigue can not be known from these results. Although the exact causes of muscle fatigue are unknown, the possible causes can be divided into central and peripheral causes. Central causes would include such things as failure of the respiratory center in the brain and failure of the impulse to propagate down the efferent nerve to the respiratory muscles. Peripheral causes would include failure of the contractile apparatus in the muscle fiber, an interruption of the depolarization of the muscle fiber membrane, and inadequate energy supplies in the muscle fiber. Any of these or other possible fatigue mechanisms could have caused the decrease in expiratory pressure noted after short term, high intensity exercise in this study.

Although this study did not attempt to differentiate between the fatigue mechanisms, it is unlikely that central causes are responsible since the fatigue lasted up to 5 minutes. With central fatigue, recovery would be fairly rapid. It is also doubtful that fatigue resulted from energy depletion. Although no measurements of feelings of fatigue were taken, trained subjects expressed feeling "fully recovered" well before the 5 minutes of recovery were finished. More research into fatigue mechanisms is needed to follow through with the current observations.

In this study, neither VO_{2max} or Ve_{max} were correlated significantly to percent decline in Pe over recovery (See Figs. 3 & 4). This is to be expected since VO_{2max} and

Ve_{max} were different between trained and untrained but percent declines in Pe were not different between groups. Since VO_{2max} was not correlated to percent decline in Pe it seems likely that having a higher VO_{2max} does not provide any protection against respiratory muscle fatigue during short term, high intensity exercise bouts. The Ve_{max} in this study refers to the maximum ventilations during the VO_{2max} test and not the exercise bout at 90% of VO_{2max}, therefore, it is not surprising that there is not correlation. Perhaps if ventilations were measured during the 90% exercise bout there would have been a more significant correlation. Although no connection was seen in this study, it is logical that the greater a person's ventilations during an exercise bout, the more they use their expiratory muscles and the more likely it will be that their expiratory muscles will fatigue.

From this study, it is concluded that an exhaustive bout of exercise at 90% of VO_{2max} can cause a reduction in Pe_{max} in both trained and untrained subjects. The relative decrease in Pe is not different between trained and untrained although the absolute Pe values are higher in trained subjects. Future studies examining the changes in muscle fiber morphology and biochemistry before and after training are needed to determine the mechanism behind any training adaptations. More study is also needed to determine if a threshold workload exists beyond which expiratory muscle fatigue occurs.

Recommendations for Future Study

Although the results of this study add some interesting information about the expiratory muscles and their fatiguability to the current literature, there are some questions still to be answered. One question that remains to be answered is, does expiratory muscle fatigue affect performance? It has been shown in this study that expiratory muscle fatigue occurs as a reduction in Pe_{max}, but that does not show how it will affect subsequent exercise performance. A way to approach this is to fatigue the expiratory muscles by resistive breathing and then look at exercise performance afterwards using time to exhaustion as the indicator.

As was stated earlier, there has been an ongoing debate over whether muscle fatigue is caused by peripheral failure or central failure. This study did not attempt to differentiate between the fatigue mechanisms. Future studies may help trace the cause of the expiratory muscle fatigue possibly by looking at an animal model using direct stimulation of the expiratory muscles to rule out central causes. It would also be helpful to monitor energy stores in the respiratory muscles as they fatigue to see if energy is the limiting factor. Much more study needs to be done in this area not only on respiratory muscles but to find the cause of general skeletal muscle fatigue.

The current study did not attempt to find any mechanism by which the expiratory muscles of trained subjects developed improved capacity to generate Pemax. Future studies may look further at the biochemical and histochemical differences in the inspiratory and expiratory muscles before and after training. Although Uribe, et al. (1992) did attempt to look at these differences in the expiratory muscles of rats, they only used a training period of 9 weeks and they used only citrate synthase as a measure of oxidative potential and found no changes. After a longer training period, some researcher found changes in the oxidative capacity of the diaphragm, but none have found any differences in the other respiratory muscles. It may be necessary to use longer periods of training or more intense training and other measures should be studied, such as glycolitic capacity and fiber type and size.

Appendix A

METHODOLOGY

DETAILED METHODOLOGY

Subjects

7 male and 3 female trained cyclists age 20-33 were used as subjects. In order to qualify for participation in this study, subjects had to be actively training by cycling 100 miles per week and been doing so for at least 3 months. Subjects were also nonsmokers with no history of chronic pulmonary disease and no recent upper respiratory infections. Subjects were screened by completing a questionnaire (see Appendix B). $FEV_{1.0}$ values were measured both before and after exercise to eliminate any subjects with present pulmonary dysfunction.

Protocol

Subjects performed a graded exercise test on a cycle ergometer in order to determine VO_{2max}. They then returned on a second day for a high-intensity cycling bout at a workload corresponding to 90% of VO_{2max}. Each test required subjects to exercise to voluntary exhaustion. Measurements of Pe_{max} were taken before each trial, immediately post-exercise and 1, 3 and 5 into recovery. Each instrument was calibrated prior to each test.

Measurement of Pe_{max}

Maximal expiratory pressure, Pe_{max}, was measured through the use of a resistance tube attached to a mouthpiece which leads into a pressure transducer (Omega Engineering, 0-250 mmHg). A small leak was present in the tube in order to prevent glottic closure. Pressure signals were amplified (Grass P511 low-level DC, 0-30 Hz) and displayed on a strip-chart recorder. Prior to each test, the transducer was calibrated by applying known pressures with a sphyngomanometer and monitoring the pen displacement on the strip chart recorder.

Subjects, while wearing a noseclip, inhaled to total lung capacity. The airway on the apparatus was closed and each subject exhaled maximally against the resistance tube. A minimum of three resting trials was obtained before each exercise bout. More than three trials were performed if significant variation exist between the second and third trials. Measurements were also taken after both exercise trials, including within twenty seconds of test termination, and 1, 3 and 5 minutes after test termination. Subjects were verbally encouraged to give a maximal effort during each trial. Pilot data reveal a high reproducibility between trials.

Measurement of $FEV_{1.0}$

Forced expiratory volume during the first second of exhalation ($FEV_{1.0}$) was measured both before and five to seven minutes after the graded exercise trial. Subjects inhaled to total lung capacity and then exhaled as forcefully as possible into a mouthpiece connected to a spirometer (Timed vitalometer, Warren E. Collins, Inc.). Three tests before and after the exercise bouts were administered on each subject, in order to screen for any unknown respiratory disorders, such as exercise induced asthma. Posttest measurements were obtained within ten minutes of the completion of the VO_{2max} test.

Graded Exercise Test

Subjects pedaled on a Monark bicycle ergometer starting at a workload of 1.0 kg at a constant rate of 50 revolutions per minute (50 Watts). The cycle ergometer was calibrated prior to each test. Workload was increased 1.0 kg (50 Watts) every two minutes until the subject was unable to continue despite verbal encouragement. At each minute heart rate, blood pressure, ventilation and expired gas fractions were determined. Measurement of VO_2, VCO_2 and VI were made by open circuit spirometry using a Parkinson-Cowen dry gas meter to measure ventilation and a Metex S-3A oxygen analyzer and a Metex CD-3A carbon dioxide analyzer to compute VO_2 and VCO_2. Subjects breathed through a low resistance non-rebreathing valve, with the expired gases passing into a 5-1 mixing chamber. The gas analyzers were calibrated before each test using standardized gases. Criteria for assessment of VO_{2max} included a respiratory exchange ratio greater than 1.05, a heart rate ± 10 bpm of age predicted maximum, and identification of a plateau in VO_2 with an increase in exercise intensity. If two of the three criteria are met, then the highest VO_2 recorded was chosen as the subject's VO_{2max}. Pe_{max} measurements was performed as described previously.

Constant workload exercise

Subjects reported to the lab on a second day in order to perform a high intensity, constant workload exercise bout. After a two minute warm-up period, subjects cycled at a workload corresponding to 90% of their VO_{2max}. Workload at 90% VO_{2max} was calculated by taking 90% of measured VO_{2max} and using the following metabolic equation to solve for workload:

$$90\%\ VO_2\,\mathrm{ml \bullet min^{-1}} = \mathrm{WL\ kg \bullet m \bullet min^{-1}} \times 2\,\mathrm{ml \bullet kg^{-1} \bullet m^{-1}} + 3.5\,\mathrm{ml \bullet kg^{-1} \bullet min^{-1}} \times \mathrm{kg(BW)}$$

(American College of Sports Medicine, 1991)

They cycled at this workload until they were unable to continue despite verbal encouragement. During this exercise trial, heart rate and blood pressure were determined. Pe_{max} measurements were performed as described previously.

Statistics

To determine the effects of exercise protocol and measurement period on Pe_{max}, a two-way ANOVA, adjusted for repeated measures for time was performed. Where indicated, a Duncan's post-hoc exam was used to determine differences between time periods. Also, a two way ANOVA adjusted for repeated measures for time was used to examine the differences between trained and untrained. The data on untrained individuals are form a study in which untrained individuals completed the same protocol as the trained subjects in this study.

Appendix B

SUBJECT QUESTIONNAIRE

Questionnaire

Name ____________________________

Date________________

Age __________

Subject number___________

Height ____________ Weight_______________

1. Have you had any recent illness, such as the flu or any respiratory infections?

2. Do you suffer from any respiratory disorders, such as asthma, that you know of?

3. Do you smoke?

4. Do you currently have any medical problems that would limit you from participating in high intensity exercise?

5. Do you have any orthopedic problems that would impair your ability to ride a stationary bike?

6. Approximately how many miles a week do you run or ride? For how long have you been maintaining this workout level?

Appendix C

INFORMED CONSENT AND HUMAN SUBJECT FORMS

Informed Consent for Participation in this Study

1. Explanation of the Exercise Test

You will perform an exercise test on a cycle ergometer. The exercise intensity will begin at a level you can easily accomplish and will be advanced in stages depending on your fitness level. We may stop the test at any time because of signs of fatigue or you may stop when you wish because of personal feelings of fatigue or discomfort. Additionally, you will exhale maximally through a clean mouthpiece against a resistance three times prior to exercise and four times after exercise. You will breath only room air at all times.

2. Risks and Discomforts

There exists the possibility of certain changes occurring during the test. These include abnormal blood pressure, fainting, disorders of the heart beat, and in rare instances, heart attack, stroke or death. Every effort will be made to minimize these risks by evaluation of preliminary information relating to your health and fitness and by observations during testing.

3. Responsibilities of the Participant

Information you possess about your health status or previous experiences of unusual feelings with physical effort may affect the safety and value of your exercise test. Your prompt reporting of feelings of effort during the exercise test itself are also of great importance. You are responsible to fully disclose such information when requested by the testing staff.

4. Benefits to be Expected

The results obtained from the exercise test may assist you in evaluating your training and furthering your fitness goals.

5. Inquiries

Any questions about the procedures used in the exercise test or in the estimation of functional capacity are encourage. If you have any doubts or questions, please ask us for further explanations.

6. Freedom from Consent

Your permission to perform this exercise test is voluntary. You are free to deny consent or stop the test at any point if you so desire.

I have read this form and I understand the test procedures that I will perform. I consent to participate in this test.

______________________________	______________________________
Signature of Witness	Signature of Subject

CERTIFICATE
OF
APPROVAL FOR RESEARCH
INVOLVING HUMAN SUBJECTS

Division of HPE

The Human Subjects Committee of the Division of Health and Physical Education has reviewed the research proposal of Susan Stolarski

entitled Evaluation of Maximal Expiratory Pressures Following A High Intensity Bout of Exercise in Highly Trained Individuals

The members have judged the subjects participating in the related experiment (not to be at risk) as a result of their participation.

(If a risk proposal) Procedures have been adopted to control the risks at acceptably low levels. The potential scientific benefits justify the level of risk to be imposed.

Members of Divisional
Human Subjects Committee

Chairman — 9/10/92 Date

9/14/92 Date

Date

Date

REQUEST FOR APPROVAL OF RESEARCH PROPOSAL IN THE DIVISION OF HPE

Submitted to

Elyzabeth J. Holford
Chairman, Division Human Subjects Committee and/or
Chairman, Institutional Review Board

Susan Stolarski
Principal Investigator

TITLE: Evaluation of Maximal Expiratory Pressure Following a High Intensity Bout of Exercise in Highly Trained Individuals

BACKGROUND/SCIENTIFIC JUSTIFICATION: Several studies have examined the effects of an extremely long term bout of exercise, such as a marathon, on respiratory muscle function. One study in particular found decreased expiratory pressures following a marathon (Loke, 1982). Only one published study has looked at the effects of a short term high intensity bout of exercise on expiratory pressures. Bye, et al., 1984 found no change in expiratory pressures in untrained subjects following an exercise bout at 80% of VO_{2max}. An unpublished study (Wilkins, 1991), on which this study is based, found a decrease in expiratory pressue lasting up to 5 minutes following an exercise bout at 90% of VO_{2max}. This study will help to define the limits of the respiratory system further.

PURPOSE(S): The purpose of this study is to determine the effects of a bout of exercise at 90% of VO_{2max} on maximal expiratory pressure in highly trained cyclists.

EXPERIMENTAL METHODS & PROCEDURES: 10 trained cyclists will complete a graded exercise test to voluntary exhaustion while expired gases are measured to determine VO_{2max}. The exercise test will consist of 2 minute stages and subjects will be increased 50 Watts per stage. Subjects will then return on a second day to complete a cycling bout at 90% of their VO_{2max} after a 3 minute warm-up at 50 Watts. Subjects will perform a maximal expiratory maneuver from total lung capacity at least 3 times before the ride and also immediately post exercise, 1 minute post exercise, 3 minutes post exercise, and 5 minutes post exercise.

STATEMENT DESCRIBING LEVEL OF RISK TO SUBJECTS: Since subjects will be trained cyclists who are in good physical condition, risk will be minimal.

PROCEDURES TO MINIMIZE SUBJECT RISK (IF APPLICABLE): Heart rates by EKG and blood pressures will be taken throughout both exercise bouts and sumptoms will be monitored. EXercise will be discontinued if any problems occur.

RISK/BENEFIT RATIO (IF RISK PROJECT): Since risk is minimal the information gained will be useful in examining the functioning of the respiratory system.

Appendix D

RAW DATA AND STATISTICAL ANALYSES

Summary ANOVA for Pe (mmHg) for Trained and Untrained Groups at max, IPE, 1MIN, 3MIN & 5MIN

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	57914.41	57914.41	26.86	0.0000
Error 1	20	43118.89	2155.944		
Time	4	2988.717	747.1793	16.92	0.0000
Interaction (group x time)	4	328.763	82.19074	1.86	0.1255
Error 2	80	3533.713	44.17141		
Total (Adj)	109	107884.5			

ANOVA for Pe_{max} (mmHg) for Trained and Untrained Groups

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	14012.83	14012.83	23.24	0.0001
Error	20	12056.75	602.8377		
Total (Adj)	21	26069.58			

ANOVA for Pe_{IPE} (mmHg) for Trained and Untrained Groups

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	8789.491	8789.491	24.36	0.0001
Error	20	7215.567	360.7784		
Total (Adj)	21	16005.06			

ANOVA for Pe_{1MIN} (mmHg) for Trained and Untrained Groups

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	11085.91	11085.91	25.47	0.0001
Error	20	8704.619	435.231		
Total (Adj)	21	19790.53			

ANOVA for Pe_{3MIN} (mmHg) for Trained and Untrained Groups

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	12278.59	12278.59	31.36	0.0000
Error	20	7829.836	391.4918		
Total (Adj)	21	20108.43			

ANOVA for Pe_{5MIN} (mmHg) for Trained and Untrained Groups

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	12076.28	12076.28	22.27	0.0001
Error	20	10845.87	542.2933		
Total (Adj)	21	22922.15			

Summary ANOVA for Pe/Pe$_{max}$ (mmHg) for Trained and Untrained Groups at IPE, 1MIN, 3MIN & 5MIN

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	.013877	.013877	0.53	0.4739
Error 1	20	.5209866	2.604E-02		
Time	3	7.700E-02	2.566E-02	4.81	0.0045
Interaction (group x time)	3	1.002E-02	3.341E-03	0.63	0.6006
Error 2	60	.319905	5.331E-03		
Total (Adj)	87	.9417963			

ANOVA for Pe_{IPE}/Pe_{max} (mmHg) for Trained and Untrained Groups

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	1.216E-04	1.216E-04	0.01	0.9122
Error	20	.1951639	9.752E-03		
Total (Adj)	21	.1951639			

ANOVA for Pe_{1MIN}/Pe_{max} (mmHg) for Trained and Untrained Groups

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	1.141E-03	1.141E-03	0.09	0.7688
Error	20	.2572644	1.286E-02		
Total (Adj)	21	.2584062			

ANOVA for Pe_{3MIN}/Pe_{max} (mmHg) for Trained and Untrained Groups

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	1.180E-02	1.180E-02	1.22	0.2825
Error	20	.1936088	9.680E-03		
Total (Adj)	21	.2054154			

ANOVA for Pe_{5MIN}/Pe_{max} (mmHg) for Trained and Untrained Groups

Source	DF	Sum-Squares	Mean Square	F-Ratio	Prob>F
Group	1	1.079E-02	1.079E-02	1.11	0.3052
Error	20	.1950079	9.750E-03		
Total (Adj)	21	.2058069			

Duncan's Range Test for Trained and Untrained
Absolute Expiratory Pressure Pe (mmHg)

Summary Results = .05

Code(Time)	Mean	ABCDE
A(IPE)	72.52819	*SSSS
B(1MIN)	78.33364	S***S
C(3MIN)	79.54773	S***S
D(5MIN)	79.85864	S***S
E(REST)	88.78636	SSSS*

Duncan's Range Test for Trained and Untrained
Relative Change in Pe (Pe/Pe_{max})

Summary Results = .05

Code(Time)	Mean	ABCD
A(Pe_{IPE}/Pe_{max})	.8213167	*SSS
B(Pe_{1MIN}/Pe_{max})	.8839622	S***
C(Pe_{5MIN}/Pe_{max})	.8854204	S***
D(Pe_{3MIN}/Pe_{max})	.8966638	S***

DESCRIPTIVE DATA FOR TRAINED AND UNTRAINED SUBJECTS

subj	sex	age (yrs)	wt (kg)	Ve_{max}	VO_{2max} (ml/kg/min)	90% WL (Watts)	riding time (minutes)
1	M	25	79.0	115.87	62.37	350	5:30
2	M	20	64.0	92.24	68.76	300	3:15
3	M	29	79.0	113.83	60.48	330	13:00
4	F	23	68.0	79.24	52.60	240	5:30
5	M	23	59.5	69.21	56.24	240	10:00
6	F	28	44.0	66.72	65.18	210	6:00
7	M	23	72.5	91.98	63.67	320	7:20
8	M	21	73.5	122.46	67.87	350	6:10
9	F	32	54.5	61.01	52.60	200	3:10
10	M	22	72.5	125.74	68.23	350	4:00
x		24.6	66.65	93.83	61.80	289	6:32

PILOT DATA

Pe_{max} (mmHg) - trial sets average 3 trials

Subject	Day	Trial set 1	Trial set 2	Trial set 3
1	1	97.3	102.0	103.5
1	2	105.4	114.0	114.5
2	1	67.7	64.0	57.3
2	2	74.3	76.0	82.3
3	1	72.3	76.3	68.5
3	2	70.3	74.0	71.5
4	1	77.0	79.7	63.0
4	2	72.3	70.3	65.8
5	1	50.3	74.0	73.5
5	2	65.0	70.0	66.0
6	1	62.3	56.0	86.8
6	2	59.3	88.0	86.0

RAW DATA FOR EXPIRATORY PRESSURES (mmHg)

subj	group	Pe_{max}	PeIPE	Pe1MIN	Pe3MIN	Pe5MIN	VO_{2max}
1	1	196.15	140.38	167.31	159.62	173.08	61.43
2	1	88.24	74.51	92.16	86.27	80.39	68.76
3	1	136.54	111.54	115.38	119.23	121.15	60.48
4	1	98.08	75.00	84.62	94.23	94.23	52.60
5	1	103.85	76.92	90.38	101.92	96.15	56.24
6	1	133.33	115.69	109.80	107.84	109.80	65.18
7	1	106.25	71.88	96.88	100.00	81.25	63.67
8	1	100.00	88.24	105.88	103.92	107.84	67.87
9	1	103.85	105.77	92.31	98.08	100.00	52.60
10	1	98.04	84.31	74.51	86.27	88.24	68.23
x	1	116.43	94.42	102.92	105.74	105.21	61.71
11	2	79.40	67.65	67.65	64.71	70.59	35.00
12	2	88.24	73.53	67.65	67.65	79.41	39.00
13	2	71.88	43.75	59.38	59.38	60.16	53.50
14	2	56.25	51.56	49.22	52.34	49.22	39.00
15	2	51.47	44.12	44.12	47.06	38.24	32.00
16	2	40.63	26.56	25.78	26.56	25.78	30.00
17	2	66.18	57.35	57.35	61.76	61.76	30.00
18	2	66.25	51.88	60.00	63.13	62.50	36.70
19	2	63.75	57.50	75.00	60.00	60.00	51.00
20	2	94.12	82.35	86.76	101.47	97.06	50.00
21	2	46.09	42.19	45.31	35.16	34.38	44.00
22	2	64.71	52.94	55.88	60.29	58.82	40.30
x	2	65.75	54.28	57.84	58.29	58.16	40.00

#1-10 = TRAINED

#11-22 = UNTRAINED

CHANGES IN EXPIRATORY PRESSURES EXPRESSED AS A RATIO OF Pe DURING RECOVERY DIVIDED BY Pe_{max}.

Subj	group	Pe_{max}	Pe_{IPE}/Pe_{max}	Pe_{1MIN}/Pe_{max}	Pe_{3MIN}/Pe_{max}	Pe_{5MIN}/Pe_{max}
1	1	196.15	.7157	.8530	.8138	.8824
2	1	88.24	.8444	1.0444	.9777	.9110
3	1	136.54	.8169	.8450	.8732	.8873
4	1	98.08	.7647	.8628	.9607	.9607
5	1	103.85	.7407	.8703	.9814	.9259
6	1	133.33	.8677	.8235	.8088	.8088
7	1	106.25	.6765	.9118	.9412	.7647
8	1	100.00	.8824	1.0588	1.0392	1.0784
9	1	103.85	1.0185	.8889	.9444	.9629
10	1	98.04	.8600	.7600	.8799	.9000
x	1	116.43	.8188	.8919	.9220	.9095
11	2	79.40	.8521	.8520	.8150	.8890
12	2	88.24	.8333	.7667	.7667	.8999
13	2	71.88	.6087	.8261	.8261	.8370
14	2	56.25	.9166	.8750	.9305	.8750
15	2	51.47	.8572	.8572	.9143	.7430
16	2	40.63	.6537	.6345	.6537	.6345
17	2	66.18	.8666	.8666	.9332	.9332
18	2	66.25	.7831	.9057	.9529	.9434
19	2	63.75	.9020	1.1765	.9412	.9412
20	2	94.12	.8749	.9218	1.0781	1.0312
21	2	46.09	.9154	.9831	.7629	.7459
22	2	64.71	.8181	.8635	.9317	.9090
x	2	65.75	.8235	.8774	.8755	.8652

#1-10 =TRAINED

#11-22 = UNTRAINED

RAW DATA FOR $FEV_{1.0}$ (ml) VALUES FOR TRAINED SUBJECTS

Subject	Before exercise	After exercise
1	4.6	4.6
2	3.8	3.9
3	4.4	4.3
4	3.7	3.8
5	3.3	3.3
6	2.7	2.8
7	3.8	3.8
8	3.7	3.8
9	3.0	2.9
10	5.0	5.0

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VITA

Susan Marie Stolarski was born in Ann Arbor,MI on June 18, 1967. She is the only daughter of Dr. Richard and Shirley Stolarski. Her father is a research physicist with NASA and her mother is a mathematician with NSA. Her family moved to Crofton, MD when she was nine and she grew up there along with her younger brother, Steven. Upon her graduation from Arundel High School in 1985, she entered Virginia Tech in Blacksburg, VA. She received her Bachelor of Science degree in Health Education in May, 1989. Susan then entered the Master's program in Exercise Physiology at Virginia Tech the following August. In the summer of 1991 she became a certified Exercise Specialist with the American College of Sports Medicine. While a graduate student, she worked for the University in the Computer Science department. She also worked briefly for Weusthoff Hospital in Rockledge, FL in 1991 and 1992 as an exercise specialist. During her studies at Virginia Tech, she became interested in clinical medicine and plans to pursue a career in medicine. On December 26, 1992 she will be married to Debanjan Datta.

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