THE EFFECT OF GRADED AND SHORT-TERM, HIGH-INTENSITY EXERCISE ON EXPIRATORY MUSCLE PERFORMANCE

by

Lisa Jacqueline Wilkins

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APPROVED:

Dr. Jay H. Williams, Chairman

Dr. Charles R. Baffi

Dr. Don R. Sebolt

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(Abstract)

Lisa J. Wilkins

A growing body of research suggests that the respiratory system may be limited in its ability to meet the demands of increased ventilitory work. This is supported by studies reporting altered contractile properties of the diaphragm in response to increased ventilitations. In order to determine if expiratory muscle function is affected by increased ventilitory demand, this study evaluated maximal expiratory pressure, PeMax, in response to two separate short-term, high-intensity exercise trials. Males (n=7) and females (n=5) not currently in active physical training underwent a VO_2 max test and a constant workload of 90% of VO_2 max. PeMax was measured at rest, immediately post exercise, and at one two and five minutes into recovery for both exercise trials. PeMax values were found to decrease 12% and 17% in response to graded and constant workload conditions respectively ($P<.05$), and this decline persisted throughout the five minute recovery. No significant relationship was found between magnitude of decline in PeMax and VO_2 max or decline in PeMax and Ve max. These findings suggest that maximal graded exercise as well as exhaustive constant workload exercise corresponding to 90% of VO_2 max provide the ventilitory stimulus great enough to result in a decline in expiratory performance.

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Chapter I

INTRODUCTION

The physical limits of human performance have always been of interest. This is evident by the number and variety of sporting events which test these physical limits. The physiological processes behind these physical limitations are important not only from the standpoint of the athlete trying to maximize performance, but also in order to identify abnormal conditions in the clinical setting. Without a clear understanding of the normal condition, it is difficult to identify or fully understand the abnormal condition.

Maximal oxygen consumption, (VO_2 max), is considered the primary limitation on aerobic capacity in man. The ability to adequately transport oxygen and utilize it in the locomotor skeletal muscles is thought to be the ultimate factor which limits aerobic performance. Therefore, factors such as stroke volume, cardiac output, and oxidative capacity of the locomotor skeletal muscles are looked upon as the "weak links" in human performance.

Recent studies suggest that there may be other "weak links" within the pulmonary system which may limit it's ability to perform maximally. The pulmonary system is a highly efficient system which can readily adjust to substantial workloads effectively. Added demand on the

pulmonary system causes an increase in the work of the respiratory muscles, which may result in an increase in the force of their contraction. The increased work of the respiratory muscles expand the chest wall, creating the pressure necessary to increase tidal volume. Feedback mechanisms, such as chemoreceptors, which can detect the decline in pH or arterial oxygen saturation associated with exercise, and mechanoreceptors located on the chest wall, all work to regulate the activity of the respiratory muscles, in order to ensure that the pulmonary system is functioning as efficiently as possible.

Current research suggests that despite the capabilities of the pulmonary system, there may be a limit to the amount of work that the respiratory muscles can perform in order to meet increased metabolic demands. Research examining the inspiratory muscles during resistive loaded breathing, (Mador and Acededo, 1991) as well as studies looking at inspiratory muscles during endurance exercise, (Bye et al, 1984) suggest that the respiratory muscles reach a point at which thay are unable to meet the demands of higher workloads.

STATEMENT OF THE PROBLEM

Researchers investigating the pulmonary system as a source of limitation on human performance have largely focused their efforts on the inspiratory muscles. These individuals

have demonstrated limitations in meeting the increased metabolic demands necessary during resistive, loaded breathing as well as exercise. Studies involving the inspiratory muscles have reported significant declines in inspiratory pressures generated after loaded breathing, long term endurance exercise, as well as short-term, high-intensity bouts . This decline in inspiratory pressure has been shown to alter inspiratory muscle performance, (Mador and Acevedo, 1991). Conversly, very little has been done to investigate the possibility of expiratory muscle fatigue.

Since force development and length of the muscles cannot be measured directly, expiratory muscle performance is characteristically evaluated by measuring maximum expiratory pressure (Pemax), (Rochester et al., 1979). PeMax, a measurement of the overall strength of the expiratory muscles is considered the optimal measurement to determine muscle performance. Studies using expiratory resistvie loaded breathing as a stressor on the expiratory muscles have demonstrated that a point is reached at which maximum expiratory pressure declines. Suzuki et al., (1991), reported a significant decrease in Pemax for up to sixty minutes after loaded expiratory breathing, In this study, loaded breathing caused a decline in breathing frequency and an increase in tidal volume, which is consistant with other studies of this nature (Garrard and Lane, 1978). This suggests that the

expiratory muscles fatigue as a result of increased resistance rather than increased respiratory rate.

Although resistive breathing studies suggest that expiratory muscles fatigue in response to increased workloads, the question remains as to whether increased respiratory rates, such as those seen during exercise, could ever become a cause of fatigue in the expiratory muscles resulting in a limitation of VO_2 and subsequent exercise performance. Studies analyzing maximal expiratory pressures in runners, bofore and after a marathon have reported a significant decline in pressure with a prolonged exercise bout (Loke et al., 1982). This indicates that prolonged exercise requires ventilitory demands great enough to limit expiratory muslce function in these athletes. It is unknown, however, whether ventilitory demands could ever become great enough during short-term, high-intensity exercise to cause significant respiratory fatigue. Although this type of exercise has been reported to decrease inspiratory pressures (Bye et al., 1984), it is not clear if expiratory pressures decline.

The purpose of this study was to determine whether a short-term, high-intensity exercise bout and graded exercise bout would elicit significant changes in Pemax to the muscles of expiration.

This study evaluated the maximum expiratory pressures generated during two different high intensity bouts.

Measurements of PeMax were compared before and after both maximal graded exercise, as well as after a short-term, high-intensity bout. This protocol allowed for comparisons in expiratory pressure generation which could indicate whether there was a significant change in muscle performance.

SIGNIFICANCE OF THE STUDY

Results of this study could help to provide more information as to what conditions are necessary to compromise expiratory muscle performance. Previous studies have suggested that exercise at high levels of VO_2 for extended periods of time, such as that seen in marathon runners may cause ventilitory demands great enough for expiratory muscle performance to decline. This investigation allowed expiratory muscle performance to be evaluated prior to and following separate short duration exercise bouts at a greater percentage of VO_2 max, as well as graded exercise in subjects who are not endurance athletes. This could help to determine whether an intense, exhaustive bout is sufficient to fatigue the expiratory muscles or if a more extended stimulus is necessary.

RESEARCH HYPOTHESES

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

1. Ho: There was no change in Pemax following a graded exercise test to exhaustion

2. Ho: There was no change in Pemax following an exhaustive constant workload bout at 90% of VO_2 max.

DELIMITATIONS

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

1. The subject population was delimited to male (N=7) and female (N=5) age 19 to 24 years, who were apparently healthy, nonsmokers not currently in active physical training.

2. The evaluation of expiratory muscle performance in this study was delimited to the measurement of maximum expiratory pressure (PeMax).

3. This evaluation was delimited to the muscles of expiration.

4. This investigation was delimited to two short-term, high-intensity conditions, including maximal graded exercise, and constant workload exercise corresponding to 90% VO_2 max.

5. The results of this study are delimited to the ability of the expiratory muscles to generate maximum pressure.

LIMITATIONS

The investigatory acknowledged the following limitations:

1. Due to the specific population evaluated in this study,

the results are limited to apparently healthy college age individuals not in active physical training.

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. It was assumed that changes in Pemax were due to changes in the function of the expiratory muscles.
4. It was assumed that no significant changes in VO_2 max occurred between the two conditions.

DEFINITIONS AND SYMBOLS

Active physical training- Performing steady state aerobic exercise for a period of at least twenty minutes, three days a week

Forced expiratory volume ($FEV_{1.0}$)- The volume of air that can be forceably exhaled in the first second after a maximal inhalation.

Maximum expiratory pressure (PeMax)- The maximum amount of pressure that can be generated after a maximal inhalation.

Maximum inspiratory pressure (PiMax)- The maximum amount

of pressure that can be generated after a maximal exhalation.

Tidal volume- The volume of gas inspired or expired during each respiratory cycle.

Residual volume- The volume of gas remaining in the lungs at the end of a maximal expiration.

Vital capacity- The largest volume measured on complete expiration after the deepest inspiration without forced or rapid effort.

SUMMARY

The pulmonary system is considered to be a highly efficient system which is able to meet increased metabolic demands effectively. However, recent studies suggest that a point may be reached at which the respiratory muscles are unable to meet the demands placed on them. It has been demonstrated that inspiratory muscle performance declines in response to an increased resistance, (Mador and Acevedo, 1991) as well as a prolonged, (Bye et al.) or high ventilitory stimulus, (Mador and Acevedo, 1991).

It has been hypothesized that there may be limitations in expiratory muscle function as well. Studies evaluating the maximum pressure generated by the expiratory muscles have reported declines in pressure after resistive, loaded breathing, (Suzuki et al., 1991), as well as after prolonged ventilitory demands, such as those associated with marathon

running, (Loke et al, 1982). It is unknown, however, if a short-term, high-intensity type of exercise could cause ventilitory demands great enough to alter expiratory pressure. The purpose of this study was to evaluate the maximum expiratory pressure generated during both a maximal graded exercise test and a short-term, high-intensity exercise bout to provide insight as to whether this type of stimulus could alter expiratory muscle performance.

Chapter II

REVIEW OF LITERATURE

Recent literature evaluating maximal exercise performance has implicated the respiratory system as a possible source of limitation at high exercise intensities. In order to identify possible limitations on human performance, this review evaluates the ability of the respiratory system to adequately meet the increased demands associated with obstructive disease, external loaded breathing and exercise. This review focuses on the muscles of respiration, both the inspiratory and expiratory muscles, and their fatigue as a possible source of limitation on exercise performance.

RESPIRATORY SYSTEM

The pulmonary system is considered to be a highly efficient system which effectively meets the added metabolic demands of steady-state exercise. Characteristically, factors such as cardiac output and oxidative capacity of the locomotor skeletal muscles have been thought to limit human performance rather than the pulmonary system's ability to adequately oxygenate the blood. This is supported by research which indicates that at maximal exercise, maximal ventilitory volumes are not reached, ventilation to perfusion ratios are almost uniform, and diffusion capacity of the lung is in

excess of that required to maintain hemoglobin saturation of end-pulmonary capillary blood (Shepherd, 1958; Bryan et al., 1964 and Dempsey et al., 1977).

However, some research in this area suggests that some of the above pulmonary "reserves" may actually be surpassable and that information regarding the homeostatic regulation of these capacities may be inaccurate (Dempsey et al., 1984). It is well established that alveolar to arterial oxygen partial pressure difference widens linearly in response to oxygen uptake, (Asmussen and Nielson, 1960) and that arterial to venous oxygen content widens as well, (Dempsey et al., 1977) suggesting the possiblility of an eventual limit on oxygen transport. In addition, a few studies have reported significant reductions in arterial oxygen partial pressures as well as oxygen saturation in highly trained athletes working at high intensities (Rowell et al., 1964, and Gledhill et al., 1980). These findings suggest the possibility of a theoretical limit to the capabilities of the pulmonary system.

Dempsey et al., (1984) observed that the typical hyperventilitory response observed during high intensity exercise was not seen in highly trained individuals who were exercising at extremely high intensities. This hyperventilation, commonly seen in response to metabolic acidosis, hypoxemia and exercise was reported to be absent, minimal, or uncorrelated with the magnitude of metabolic

acidosis or hypoxemia at these very high workloads. Dempsey et al. (1984), attributed these findings to mechanical limitations present within the pulmonary system during periods of increased demand. This idea is further supported by a study which implemented helium, a gas less dense than nitrogen, breathing to reduce the mechanical work required by the muscles of respiration in trained subjects at high-intensity exercise (Nattie and Tenney, 1970). Nattie and Tenney (1970) reported an increased hyperventilitory response to high intensity exercise with helium breathing suggesting that the muscles of respiration may be unable to keep up with the added demands placed on them during periods of increased work.

These studies strongly suggest that a limit is reached regarding the respiratory system's ability to meet the metabolic demands associated with high intensity exercise. They suggest that mechanical factors ultimately prevent the respiratory system from keeping pace with increased exercise intensities, thus limiting exercise performance. Since mechanical factors are thought to play an important role in this limitation of respiratory system performance, respiratory muscle fatigue was examined in this review.

INSPIRATORY MUSCLE PERFORMANCE

The muscles of inspiration, which include the external intercostals, internal obliques, and diaphragm, are

responsible for approximately two thirds of the work associated with respiration. Upon contraction, the inspiratory muscles increase the thoracic volume which decreases thoracic pressure, causing air to flow into the lungs. Since these muscles are constantly in use, they have great oxidative capacity, which predisposes them to meet metabolic demands effectively (Dempsey, 1984). However, recent investigations have reported that under some conditions a point is reached at which these muscles are unable to keep up with the demands placed on them. The result is a decline in inspiratory pressure and, in some cases, a subsequent decline in exercise performance (Mador and Acevedo, 1991).

Chronic Obstructive Pulmonary Disease (COPD)

Inspiratory muscle performance was first evaluated in patients with advanced chronic obstructive pulmonary disease, in an effort to determine the role that the inspiratory muscles play in respiratory failure. Respiratory failure is a complex phenemenon in which numerous factors interact to ultimately cause failure; a phenomemon which encompasses hypoxemia and acidemia, hyperinlflation of the lungs, impaired respiratory drive and increased resistance to airflow. Several investigators have documented respiratory fatigue in this disease, as evidenced by marked reductions in PiMax (Braun and Rochester, 1977), as well as diaphragmatic and

intercostal electromyographic changes (Cohen et al, 1982). Decreases in Pimax have been shown to correspond with hypoventilation and hypoxemia which suggest that inspiratory muscle fatigue plays an important role in the respiratory failure which occurs in the later stages of COPD. (Cohen et al., 1982). Although the exact mechansism of respiratory failure is not clear, it is hypothesized that the increased airway resistance associated with COPD places added demands on the muscles of inspiration at a time when metabolic factors such as hypoxemia and acidemia cause the muscles to work inefficiently. Mechanical factors also seem to play a role in inspiratory muscle fatigue and subsequent respiratory failure. Braun and Rochester, (1977) reported a high correlation between progressive shortening of the diaphragmatic muscle as measured roentgenographically, and decreases in PiMax in COPD patients. Furthermore, mechanical breathing abnormalities such as respiratory alterans and abdominal paradox are widely observed in patients with respiratory failure and is considered a reliable clinical sign of inspiratory muscle fatigue (Cohen et al, 1982). All of these factors interplay to result in inspiratory muscle fatigue, which results in a downward spiral towards respiratory failure (Rochester et al., 1987).

External loaded breathing

In order to determine if inspiratory muscle fatigue could be induced in apparently healthy individuals, a model of external resistive breathing was devised to mimic the resistance experienced in COPD patients. By analyzing transdiaphragmatic pressures as well as electromyogaphic patterns of the diaphragm, it was reported that the diaphragm could be fatigued using external loaded breathing in healthy individuals (Roussos and Macklem, 1977). Roussos and Macklem (1977), estimated that the threshold for diaphragmatic fatigue was a pressure equal to 40% of maximum pressure generated. They observed that the diaphragm could generate pressures under this threshold indefinitly, but pressures greater than 40% of maximum pressure would result in eventual fatigue. This is supported by Gross et al., (1979), who report that at 25% of maximum diaphragmatic pressure, a fatiguing pattern was not seen upon electromyographic analysis, but a pattern of fatigue was reported at 50% and 75% of maximum diaphragmatic pressure.

In contrast to the previously mentioned studies, Bellmare and Grassino (1982), reported an electromyographic pattern of fatigue at a workload of 15% of maximum diaphragmatic pressure. The reason for the discrepancy between investigations may be attributed to the inspiratory time. Bellmare and Grassino (1982) discovered that the ratio of inspiratory time to the time of the total tidal cycle has an

impact on the rate of fatigue in the diaphragm. They determined that the greater the inspiratory time in relation to the total tidal cycle, the earlier the onset of inspiratory muscle fatigue. This would explain the descrepancies between the study by Gross et al. (1979), which reported a ratio of .6, and the investigation by Bellmare and Grassino (1982), which reported a ratio of 1.0. Further investigation revealed that this ratio was different in the conflicting studies (Bellmare and Grassino, 1982).

Although the immediate physiological response to loaded breathing is highly variable, (Axen and Sperber-Haas, 1979; Read et al, 1974), prolonged reponse to steady-state loaded breathing is well established. It has been widely reported that the inspiratory muscles work to preserve tidal volume by a variety of possible mechanisms (Hof et al., 1986). Although the primary mechanism behind this preservation of tidal volume has not yet been identified, an increase in the duration of inspiration, a decrease in the frequency of breathing and a slight decrease in ventilation have all been widely observed (Hof et al, 1986 and Axen et al, 1983). Ironically, a prolongation of inspiratory time to preserve tidal volume appears counterproductive in that it is not the most efficient way to maintain mean inspiratory pressure and therefore minimize the cost of breathing (Rochester and Bettini, 1976 and Younes and Riddle, 1984). However, this mechanism allows

for a slight decrease in ventilation which may be important in the reduction of sensory disturbances associated with the load. (Hof et al., 1986).

Studies evaluating inspiratory muscle performance in COPD patients as well as external loaded breathing trials indicate that the muscles of inspiration do indeed fatigue in response to an increased workload. Declines in Pimax seen in association with hypoventilation and hypoxemia in COPD patients suggest that inspiratory muscle fatigue is an important factor in the mechanism of respiratory failure. In addition, external loaded breathing studies indicate that this fatigue occurs in apparently healthy populations as well, with the threshold for fatigue dependant upon the pattern of ventilation.

Exercise Studies

Since the pattern of ventilation seems to play an important role in inspiratory muscle fatigue with external loaded breathing studies, the question arises as to situations where ventilitory demands are increased dramatically such as during exercise. The importance of preserving ventilation during loaded breathing could suggest that an increased ventilitory demand could be fatiguing to the inspiratory muscles, and may subsequently affect exercise performance.

Research in the past has ruled out the idea that increased ventilitory demands during exercise place a limitation on the respiratory muscles and subsequent exercise performance because of investigations that suggested that the pulmonary system did not reach its maximal capacity during high intensity exercise. This is supported by the following investigations. First, Shephard et al, (1967), demonstrated that during maximal exercise, subjects were able to voluntarily increase minute ventilation, suggesting that maximal ventilations were not reached during maximal exercise, and a large respiratory reserve prohibited the respiratory muscles from ever becoming a limiting factor on exercise performance. Second, Hughes et al., (1968), in an effort to determine the limiting factors at maximal exercise, compared cardiovascular, respiratory, and metabolic adaptations to increased workloads under various concentrations of inspired oxygen. They reported that maximal ventilitory volume is not normally reached in high intensity exercise.

Although these studies suggest that maximal ventilations are not reached at high levels of exercise, recent research suggests that the ventilitory demands experienced during exercise may be great enough to fatigue the inspiratory muscles. Although maximum ventilitory values are not acheived, prolonged periods of time at a high percentage of maximum ventilitory values may result in fatigue. A study by Loke et

al., (1982) reported a 16.5% decline in PiMax, as well as a 19.7% decline in transdiaphragmatic pressure at inspiratory capacity following a marathon race. This decline in performance of the inspiratory muscles suggests that this endurance exercise caused a certain amount of fatigue to the muscles of inspiration. This is further supported by a study by Fregosi et al. (1987), which reported a significant increase in intracellular lactate levels in rat diaphragm after running to exhaustion. In addition, post exercise MVV measures have been shown to be significantly lower than controls both after a 60 minute exhaustive running bout and a marathon (Loke et al., 1982).

Although MVV values have been reported to decline after marathon running and other long term exhaustive exercise, short-term, high-intensity exercise does not appear to elicit a decline in this parameter (Bender and Martin, 1984). This raises the possibility that a short, intensive exercise stimulus may not provide a ventilitory demand great enough to cause fatigue of the inspiratory muscles. In contrast, Coast et al., (1990) reported a 17% decline in PiMax after maximal graded exercise. This is further supported by Bye et al., (1984), who reported a decline in Pdi as well as a 20% decline in the ratio of high to low frequency components of the diaphragm EMG after a short-term, high-intensity constant workload exercise bout. This suggests that this exercise

stimulus was indeed great enough to evoke diaphragm fatigue. Bye et al., (1984) also found that substituting room air with 40% oxygen reduced ventilation, and delayed the onset of diaphragmatic fatigue. This indicates that the ventilitory demand plays a primary role in the fatigue of the inspiratory muscles during short-term exercise.

Effect on Subsequent Performance

An important question which arises from the previous studies reporting inspiratory muscle fatigue, is the impact that this fatigue has on exercise performance. Recent investigations suggest that the declines in inspiratory pressures reported may cause a limitation on subsequent exercise performance. In other words, the changes seen in inspiratory muscle performance may actually result in a limitation on an individual's exercise capacity. For example, Mador and Acevedo (1991), had subjects inspire against a load equivalent to 80% of their maximum mouth pressure until they were unable to generate this target pressure. Subjects then cycled at 90% of their VO_2 max until volitional exhaustion. Following loaded breathing, exercise time was decreased, as well as VO_2 and heart rate during the last minute of exercise. Additionally, ventilation and subjective ratings of respiratory effort were greater after the induction of fatigue. This supports earlier findings by Martin et al.

(1982), reporting that individuals who were subjected to prior ventilitory work, (150 min of sustained MVV) experienced a decline in time to exhaustion during short-term maximal running. Subjects stopped at a lower ventilation, heart rate and VO_2 after prior ventilitory work. These investigations demonstrate that inspiratory muscle fatigue results in a decline in subsequent exercise performance and may be an important limiting factor on human performance.

Trained versus Untrained Subjects

In light of research reporting that PiMax and Pdi decline in response to increased ventilitory demand and that this decrease in pressure may have a negative impact on exercise performance, efforts have been made to determine whether this affects trained and untrained individuals equally. Dempsey (1986), hypothesized that a greater decrease in inspiratory muscle performance would be observed in highly trained individuals. He reasoned that trained individuals had acquired the adaptations in VO_2 max and skeletal muscle oxidative capacity necessary to reach ventiliations high enough to limit the capabilities of the pulmonary system. However, a recent study by Coast et al (1990), demonstrated that untrained subjects showed significant declines in PiMax for up to two minutes following maximal exercise while a group of highly trained cross country skiers showed no significant

declines in PiMax. Furthermore, the 12% decrease in PiMax found in the untrained subjects in this study are similar to declines seen in a similar study by Bye et al. (1984), using subjects who were not in active physical training.

One reason for the lack of inspiratory muscle dysfunction in the cross country skiers in Coast's study may be due to a training effect on the muscles of inspiration. Several investigations suggest that a training effect on the muscles of respiration is possible. Three pieces of evidence support this idea. First, respiratory muscle training techniques have been shown to increase maximal sustained ventilation, (Bradley and Leith, 1978 and Morgan et al., 1987), as well as pressure generating capacities of the respiratory muscles (Leith and Bradley, 1976). However, the benefits accrued were very specific to the type of training used and did not result in an increase in exercise capacity. Second, artificially increasing the work of breathing has been reported to result in a training effect of the respiratory muscles. Farkas and Roussos, (1974) as well as Keens et al., (1978) reported increases in marker enzymes for aerobic metabolism in the diaphragm of animals with artificially-induced airway resistance. This suggests that the diaphragm did indeed undergo a training effect in response to an increased work of breathing. Third, some studies have reported increases in the level of diaphragm succinate dehydrogenase (Leiberman et al.,

1972) and other enzymes of beta oxidation (Moore and Gollnick, 1982; Ianuzzo et al, 1982) in response to dynamic endurance exercise training, demonstrating that whole body exercise may also bring about a training effect of the respiratory muscles. However, others have found no change in enzymatic composition of the diaphragm after high-intensity exercise training (Metzger and Fitts, 1986), suggesting that endurance training may be more effective in eliciting a training effect.

EXPIRATORY MUSCLE PERFORMANCE

Although many investigations have examined the role of inspiratory muscle fatigue on the limitations of the pulmonary system, very little research is available regarding the expiratory muscles, their role in respiratory muscle fatigue and possible effects on subsequent exercise performance.

The muscles of expiration, which include the internal intercostals, rectus abdominus, external and internal obliques and transversus abdominus, do not contribute to respiration during periods of rest or passive breathing (Campbell and Green, 1955; Gilbert and Auchincloss, 1969). These muscles are recruited only during periods of increased respiratory work in an effort to preserve functional residual capacity (FRC). Electromyographical analysis has demonstrated that the muscles of expiration become actively recruited at ventilations of 40 L/min (Campbell and Green, 1955; and Strohl

et al., 1981). This value is in agreement with investigations using other techniques for determining expiratory muscle recruitment. Grimby et al. (1976) reported a pressure volume displacement of the abdominal muscles at ventilations of 40-80 L/min, indicating that expiratory muscle recruitment was initiated at this threshold. Henke et al. (1988) reported expiratory muscle recruitment at 60 L/min by using a helium dilution technique to measure end expiratory lung volume.

Measurement of Expiratory Muscle Performance

The measurement most commonly used to assess the performance of the expiratory muscles is maximal expiratory pressure (PeMax) measured at the mouth. This measurement, obtained by acheiving a maximal expiration after inhaling to total lung capacity, is considered a simple, reproducible indicator of expiratory muscle performance. Black and Hyatt, (1969) measured PeMax in 120 subjects and reported a coefficient of variation for duplicate measurements of 9% and no significnat differences between measurements obtained over three consecutive days. The values reported by Black and Hyatt, (1969) are in argeement with other maximum expiratory pressures obtained using other techniques, including intraesophageal pressure measurements (Hyatt, as referenced in Black and Hyatt, 1969). Furthermore, the coefficient of variation reported is in agreement with results obtained from

other muscle groups tested, (Ringqvist, 1966; and Tornvall, 1963), suggesting that this is an acceptable amount of variation. Statistical analysis yeilded no significant impact on age below the age of 55 and found that the pressures obtained in females were 65%-&0% lower than those obtained by males (Blackk and Hyatt, 1969).

COPD

Expiratory muscle fatigue was first examined in patients with COPD, where respiratory muscle fatigue is thought to play an important role in respiratory failure. Several investigations evaluating inspiratory muscle performance have reported declines in inspiratory muscle strength which are believed to play an important role in the hypoventilation and hypoxemia observed in respiratory failure. Although inspiratory muscle fatigue is well documented in this disease, (Rochester et al., 1987) it is uncertain as to whether the resistive loading associated with COPD causes sufficient expiratory muscle activity to elicit fatigue. Byrd and Hyatt, (1968) report no difference in the strength of the expiratory muscles in patients with COPD compared with healthy individuals. However, in their study, they did not correct their data to reflect the abnormal static recoil seen in patients with COPD. When correction for abnormal recoil is employed (Rochester et al., 1987), declines in PeMax and FEV1

are observed, reflecting a decline in expiratory muscle performance. Furthermore, they reported that this expiratory weakness may contribute to impairment of inspiratory muscle function.

External Loaded Breathing

In order to further investigate whether an increase in the work of breathing can cause expiratory muscle fatigue, Suzuki et al., (1991) used a model of external loaded breathing to evaluate respiratory muscle strength in normal, healthy individuals. After subjects breathed against an expiratory resistance for 60 minutes declines in Pemax of 16% were observed, and these declines persisted for up to an hour after loaded breathing. Suzuki et al., (1991) further evaluated the effect of resistive loading on the expiratory muscles by performing EMG analysis on the rectus abdominus muscle. EMG analysis confirmed that the rectus abdominus was fatigued by expiratory loading.

In this study, expiratory loading resulted in a decline in breathing frequency (55%) and an increase in tidal volume (60%) which is in agreement with other studies of expiratory loading (Garrard and Lane, 1987). In addition functional residual capacity was increased presumably to increase the expiratory force generated by optimizing the pressure-volume relationship (Suzuki et al., 1991).

Exercise Studies

Although the results reported by Suzuki et al., (1991) suggest that expiratory muscle fatigue is possible in apparently healthy individuals in response to an increased workload, it does not clarify whether expiratory muscle fatigue could occur in response to an increased ventilitory demand. Inspiratory muscles have been shown to fatigue in response to incresed ventilitory demands associated with high intensity exercise which suggests that expiratory muscles may fatigue as well. Recent studies have investigated whether increased ventilitory demands such as those seen during exercise are great enough to fatigue the expiratory muscles, as they do the inspiratory muscles. Loke et al., (1982) report a 27.9% decline in PeMax in runners after a marathon race, suggesting that this endurance activity provided a ventilitory demand great enough to induce expiratory muscle fatigue. Other parameters assessing pulmonary function in marathon runners were evaluated in a study by Maron et al. Maron et al., (1979) reported a 8.6% reduction in forced vital capacity immediately after a marathon. This agrees with an early study by Gordon et al. (1924), which reports a 17% reduction in forced vital capacity after a marathon. Total lung capacity was reported to be unchanged suggesting that the decline in forced vital capacity resulted from expiratory limitations. Loke et al., (1982) reported a significnat

decline in PeMax, suggesting that expiratory muscle strength decreased after a marathon.

Very few investigations have examined expiratory muscle performance during a short-term, high-intensity exercise bout. It is still unclear as to whether this type of exercise stimulus could provide the ventilitory demands great enough to fatigue the expiratory muslces. Although relatively few studies have directly addressed this question, research in this area support the possibility that expiratory muscle fatigue may occur in response to short-term, high-intensity exercise. First, as mentioned previously, expiratory muscle recruitment has been shown to occur at rather modest exercise intensities. Ventilations of 40 L/min, which correspond to approximately 50% of VO2 max are easily surpassed during short term exhaustive exercise and can be maintained for several minutes, suggesting that the ventilitory demands may be adequate for fatigue. Second, a decline in PeMax has been reported with short-term, high-intensity exercise. O'Kroy et al., (1991) reported a significant decrease in PeMax after both a seven minute bout at 90% of VO_2 max and a thirty minute bout at 60% of VO_2 max. These values for PeMax were significantly lower than pre test values at 5, 10, and 30 minutes post exercise for both trials. Finally, forced vital capacity has been reported to decrease significantly following a short-term, high-intensity bout at 90% of VO_2 max (O'Kroy et

al., 1991). This decline in forced vital capacity is also thought to reflect a decline in performance of the expiratory muscles, resulting in an inability to exhale to residual volume.

These previously mentioned studies suggest that the muscles of expiration may fatigue during periods of increased metabolic demand. Studies evaluating expiratory muscle performance in patients with COPD, as well as healthy individuals breathing against an expiratory resistance show a decline in expiratory pressure as well as a fatiguing EMG pattern, suggesting that an increased workload causes fatigue of the expiratory muscles. Furthermore, exercise studies demonstrate that the increased ventilitory demand associated with marathon running and high-intensity exercise are great enough to elicit expiratory muscle fatigue.

SUMMARY

The objective of this review was to examine the role of the pulmonary system, or more specifically the respiratory muscles on the limitation of human performance. Both the inspiratory and expiratory muscles were reviewed, in order to determine their function during periods of increased metabolic demand, including airway obstruction, and increased ventilition.

Although the pulmonary system has characteristically been thought to have an adequate reserve to meet increased metabolic demands effectively, recent evidence suggests that this reserve may have been overestimated and that mechanical factors may ultimately limit the ability of the pulmonary system to meet the incresed demands of high intensity exercise (Dempsey et al., 1984). This is supported by studies which reported a deline in arterial oxygen saturations, (Glendhill et al., 1980) and a widening of arterial to venous oxygen partial pressures (Demspey et al., 1977) at high exercise intensities. It is further supported by an uncorrelation of compensatory hyperventilation in response to acidosis or hypoxemia in highly trained athletes during high intensity exercise (Dempsey et al., 1984).

In support of the above notion, the inspiratory muscles appear to fatigue in response to loaded breathing. This has been demonstrated in patients with COPD (Rochester et al., 1987), as well as in apparently healthy individuals in response to external loaded breathing (Roussos and Macklem, 1977). In addition, increased ventilitory demands during exercise cause a decline in inspiratory muscle performance. For example, maximum inspiratory pressures decline after marathon running (Loke et al., 1982), and during short-term, high-intensity exercise (Bye et al., 1984). Furthermore, this change in performance of the inspiratory muscles has been

reported to affect subsequent exercise performance (Mador and Acevedo, 1991).

The expiratory muscles also exhibit functional decline in response to increased workloads and ventilation. Investigations demonstrate that expiratory muscles are recrited at rather modest ventilations (Strohl et al., 1981). In addition, maximum expiratory pressures decline in reponse to increased expiratory resistance in both COPD patients, (Rochester et al., 1987) and apparently healthy individuals (Suzuki et al, 1991). This decline is also demonstrated following marathon running (Loke et al., 1984). Although very little research has been done to establish expiratory muscle fatigue in response to short-term, high-intensity exercise, declines in PeMax as well as forced vital capacity following this type of stimulus suggest that a decline in expiratory muscle performance may occur (O'Kroy et al., 1991). This is further supported by investigations reported that expiratory muscles are recruited at rather modest ventilations (Strohl et al., 1981).

Chapter III

THE EFFECT OF GRADED AND SHORT-TERM, HIGH-INTENSITY EXERCISE ON EXPIRATORY MUSCLE PERFORMANCE

Lisa J. Wilkins

Department of Health and Physical Education
Virginia Polytechnic and State University

ABSTRACT

A growing body of research suggests that the respiratory system may be limited in its ability to meet the demands of increased ventilitory work. This is supported by studies reporting altered contractile properties of the diaphragm in response to increased ventilitations. In order to determine if expiratory muscle function is affected by increased ventilitory demand, this study evaluated maximal expiratory pressure, PeMax, in response to two separate short-term, high-intensity exercise trials. Males (n=7) and females (n=5) not currently in active physical training underwent a VO_2 max test and a constant workload of 90% of VO_2 max. PeMax was measured at rest, immediately post exercise, and at one two and five minutes into recovery for both exercise trials. PeMax values were found to decrease 12% and 17% in response to graded and constant workload conditions respectively ($P<.05$), and this decline persisted throughout the five minute recovery. No significant relationship was found between magnitude of decline in PeMax and VO_2 max or decline in PeMax and Ve max. These findings suggest that maximal graded exercise as well as exhaustive constant workload exercise corresponding to 90% of VO_2 max provide the ventilitory stimulus great enough to result in a decline in expiratory performance.

INTRODUCTION

When evaluating possible limitations on exercise performance, the pulmonary system has largely been overlooked. Due to research indicating that at maximal exercise, maximal ventilitory volumes are not reached, and ventilation to perfusion ratios are almost uniform, the pulmonary system has conventionally been considered to have more than adequate reserves to meet the demands of maximal exercise effectively (Shepherd, 1958; Bryan et al., 1964). However, recent investigations reporting reductions in arterial oxygen partial pressures as well as declines in oxygen saturation in highly trained athletes during maximal exercise suggest that there may be a limit to the capabilities of the pulmonary system (Rowell et al., 1964, Gledhill et al., 1980; Dempsey et al., 1977; Williams et al., 1986).

Research examining mechanical limitations within the respiratory system during periods of increased ventilation have demonstrated a decline in inspiratory muscle performance in response to marathon running as well as short-term, high-intensity exercise (Loke et al., 1982; Bye et al., 1984). In contrast, very little research is available regarding expiratory muscle function. Studies evaluating expiratory muscle performance using a model of resistive loaded breathing report declines in PeMax suggesting that expiratory muscle performance declines in response to an increase in the work of

breathing (Suzuki et al., 1991). However it is still unclear whether PeMax is affected by increased ventilitory work. Expiratory muscle performance has been reported to decline in response to marathon running (Loke et al., 1982), but it is still unclear whether short-term, high-intensity exercise provides the ventilitory stimulus great enough to effect expiratory muscle performance.

In this investigation, maximal expiratory pressure, (PeMax), was measured in order to evaluate expiratory muscle performance. PeMax was measured before and after both maximal graded exercise and short-term, constant workload exercise in order to determine whether these two short-term, intensive exercise bouts provide the ventilitory stimulus great enough to cause a decline in expiratory muscle performance.

METHODS

Subjects

Apparently healthy college aged male (n=7), and female (n=5) volunteers were used for this investigation. In order to qualify for participation in this study, individuals had to be nonsmokers, have no history of chronic pulmonary disease and have had no recent upper respiratory infections. In addition, only individuals who had not participated in any regular aerobic training for three months prior to the investigation were selected to participate. Forced expiratory

volume during the first second of expiration, (FEV_1) was measured both before and after exercise to eliminate any subjects with pulmonary dysfunction.

General Protocol

Subjects performed a graded exercise test on a cycle ergometer in order to determine VO_2 max, and returned on a second day for a short-term, high-intensity cycling bout at a workload corresponding to 90% of VO_2 max. Each test required subjects to exercise to voluntary exhaustion. Measurements of PeMax were obtained before each trial, immediate post-exercise and during recovery.

Measurement of PeMax

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

Subjects, wearing a noseclip, were first directed to

inhale to total lung capacity. The "airway" was closed and each subject was instructed to exhale maximally against the resistance tube. A minimum of three resting trials were obtained before each exercise bout. More than three trials were performed if variation existed between the second and third trials. Pilot data revealed a correlation of .9 between resting measurements. Measurements were also taken immediately post exercise (IPE) within 20 seconds of test termination), and one, two and five minutes into recovery. Subjects were verbally encouraged to give a maximal effort during each trial.

Graded Exercise Test

For the graded exercise test, males started at a workload of 50 Watts, and females at a workload of 25 Watts. Workload was increased 25 Watts every two minutes until the subject was unable to continue despite verbal encouragement. During the final 30 sec. of each stage, heart rate, blood pressure, perceived exertion, ventilation and expired gas fractions were determined. Measurement of VO_2, VCO_2, and Ve were made by the open circuit technique using a Rayfield dry gas meter to measure ventilation and a Metex S-3A oxygen analyzer and a Metex CD-3A carbon dioxide analyzer to determine 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_2 max included a respiratory exchange ratio greater than 1.15, a heart rate $\pm$ 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 were met, then the highest VO_2 recorded was chosen as the subject's VO_2 max. All subjects successfully met this criteria. PeMax measurements were taken both before and after the test as described previously.

Constant Workload Exercise

Subjects were brought into the lab on a second day in order to perform a short-term, high-intensity, constant workload exercise bout. After a two minute warm up period, subjects cycled at a workload corresponding to 90% of their VO_2 max. They cycled at this workload until they were unable to continue despite verbal encouragement. During this exercise condition, heart rate, ventilation, and expired gas fractions were determined as described above. PeMax measurements were performed after the test as described previously.

Statistics

To determine the effects of exercise protocol and measurement period on PeMax, a two-way ANOVA, adjusted for repeated measures was performed. Where indicated, a Duncan's post-hoc exam was used to determine individual differences between measurement periods.

RESULTS

Subjects cycled for a mean duration of 13 min on the graded protocol to a mean VO_2 max of 40 $\pm$ 2.3 ml/kg/min. Changes in PeMax following graded exercise are shown in Fig. 1. PeMax was found to be significantly lower immediatly post exercise, (12%), as compared to resting values. This decline in PeMax persisted throughout the five minute recovery.

PeMax was also found to decline after the constant workload, high-intensity bout (Fig. 2). Subjects cycled for a mean duration of 5.5 min. at a workload corresponding to 90% of their predetermined VO_2 max. PeMax dropped 17% from resting measurements and this decline persisted throughout the five minute recovery.

No significant difference in PeMax was found between the two conditions. In addition, no significant relationship was found between the decline in PeMax and VO_2 max (Fig. 4), or decline in PeMax and Ve max (Fig. 3).

DISCUSSION

The present study demonstrates that graded exercise and constant-load, high-intensity exercise results in a decline in expiratory muscle performance. This suggests that these two exercise protocols elicited a ventilitory stimulus great enough to affect expiratory muscle function in apparently healthy individuals. This finding is in agreement with other research in this area which reports declines in both inspiratory and expiratory muscle function in response to intensive exercise.

Studies evaluating expiratory muscle performance suggest that the expiratory muscles fatigue in response to increased ventilitory demand. Research demonstrates that although the muscles of expiration do not contribute to the work of passive breathing, these muscles are recruited at ventilations of 40 L/min (Campbell and Green, 1955; and Strohl et al, 1981). The present study found a mean maximal ventilation of 82 $\pm$ 6.5 L/min. This would indicate that prolonged activity at moderate to high ventilations may be capable of causing expiratory fatigue. This is supported by studies evaluating marathon runners after a race, which report declines in PeMax of 27.9% as well as reductions in FVC (Loke et al., 1982).

The present investigation supports the idea that expiratory muscle dysfunction may occur in response to short, intensive exercise. Although very few investigations have

examined expiratory muscle performance using this type of exercise stimulus, research in this area support the possibility that it may occur. Declines in PeMax have been reported in response to a seven minute bout at 90% of VO_2 max and significant declines in FVC have also been reported using this protocol (O'Kroy et al., 1991). The present study found a decline in PeMax following both maximal graded exercise and a constant workload bout at 90% of VO_2 max which supports previous findings (O'Kroy et al., 1991).

Comparisons with PiMax

The magnitude of decline in PeMax found in this study is similar to that seen in studies evaluating inspiratory muscle performance after exercise. Although patterns of recruitment are different between these two muscle groups, such a comparison is relevant because together they share the increased work of breathing associated with high ventilations (Dempsey et al., 1982). Bye et al., (1984) reported a 28% decline in transdiaphamatic pressure, (Pdi), as well as a 20% decline in the ratio of high to low frequency components of the diaphragm EMG after a short-term, high-intensity constant workload exercise bout. In addition, Coast et al., (1990) reported a 17% decline in PiMax in untrained subjects after maximal graded exercise. These studies are in agreement with the mean 14.5% decline in PeMax found in the present study.

Taken together, these results suggest that both inspiratory and expiratory muscle performance declines in response to short-term, high-intensity exercise, and the magnitude of decline is similar between the two muscle groups.

Critique of Methodology

The possibility exists that the declines in PeMax found in this study may be attributed to a lack of motivation to perform maximal breathing maneuvers after intensive exercise. If this were the situation, the declines in PeMax would reflect a generalized fatigue associated with the discomfort of exercise rather than fatigue of the expiratory muscles. This is unlikely due to several reasons. First, as previously mentioned, the magnitude of decline in PeMax in this study is consistent with other studies evaluating respiratory muscle function in response to short-term, high-intensity exercise. Second, the magnitude of decline in PeMax was not significantly different between the two conditions. It is unlikely that a lack of motivation would be so consistent in response to two different conditions that were of different duration on separate days. Finally, declines in PeMax persisted throughout the five minute recovery for both exercise trials. If the discomfort associated with high intensity exercise resulted in a a lack of motivation to exhale maximally, it would be expected that PeMax would return

to resting values once the subject had a chance to recover from the discomfort of exercise. Since PeMax values were still depressed five minutes into recovery, factors other than motivation most likely were causing the decline. This suggests that the declines seen in this investigation actually represent expiratory muscle dysfunction rather than a lack of motivation to perform maximally in these particular subjects.

Comparisons to other investigations

The decline in PeMax throughout recovery seen in this investigation is consistant with other studies in this area. The declines in PiMax found by Coast et al. (1990), after maximal graded exercise persisted throughout a five minute recovery. These findings are in agreement with O'Kroy et al. (1991), who reported declines in PeMax five minutes after a seven minute bout at 90% of VO_2 max and a 30 min bout at 60% of VO_2 max. Additionally, Suzuki et al., (1991) reported a decline in PeMax for up to 60 min following expiratory resistive loaded breathing but did not find a corresponding EMG pattern of fatigue. Although the mechanism behind this decline in PeMax is not known, it lends support to the idea that the declines in expiratory muscle performance may be persistant enough to affect subsequent exercise performance.

Relationship with VO_2 max and Ve max

The present study did not find a strong correlation between the magnitude of decline in PeMax and VO_2 max. This conflicts with research which found that highly trained subjects have lesser changes in PiMax following maximal exercise than untrained subjects (Coast et al., 1990), possibly due to a training effect on the respiratory muscles. One reason for a failure to see a relationship between PeMax and VO_2 max, as well as a relationship between PeMax and Ve max in this study might be due to the rather homogeneous population of untrained subjects. This present study consisted of 12 subjects with a mean VO_2 max of 40 + 2.3 ml/kg/min with a coefficient of variation of .58. In addition, none of the subjects in the present study would be considered to be trained and thus may not consistently reach ventilations high enough in their daily activities to result in a training effect of the respiratory muscles. It is possible that inclusion of trained subjects in the present study might have resulted in results similar to Coast et al., (1990).

Summary

The primary objective of this study was to evaluate expiratory muscle performance during two short-term, high-intensity exercise protocols. The declines in PeMax observed in response to both graded and constant-load exercise suggest

that this type of exercise stimulus provides the ventilitory demands great enough to cause a decline in expiratory muscle performance.

These data provide a framework for further research in order to determine if expiratory muscle dysfunction is ultimatly a limiting factor on exercise performance. This could have implications not only for the athlete, training to optimize exercise performance, but also in the diagnostic and functional assessment of VO_2 max of sedentary populations. Since greater declines in respiratory muscle performance are seen in sedentary people, respiratory system limitations may occur in this population that currently have been attributed to limitations in VO_2 max. In addition, recent research in the area of respiratory muscle function suggests that a training effect on the muscles of respiration may be possible in response to repeated exposure to increased ventilations. If this were to be confirmed, athletes may alter their training to optimize these potential benefits. This also may have implications for exercise training in patients with COPD and efforts to improve their ability to perform activities of daily living.

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LIST OF CAPTIONS

Figure

1 PeMax vs. Measurement Period; Incremental Trial
* $p<0.05$

2 PeMax vs. Measurement Period; 90% VO_2 max Trial
* $p<0.05$

3 Percent Decline PeMax measured after 90% VO_2 max vs. Ve max

4 Percent Decline PeMax measured after 90% VO_2 max vs. VO_2 max

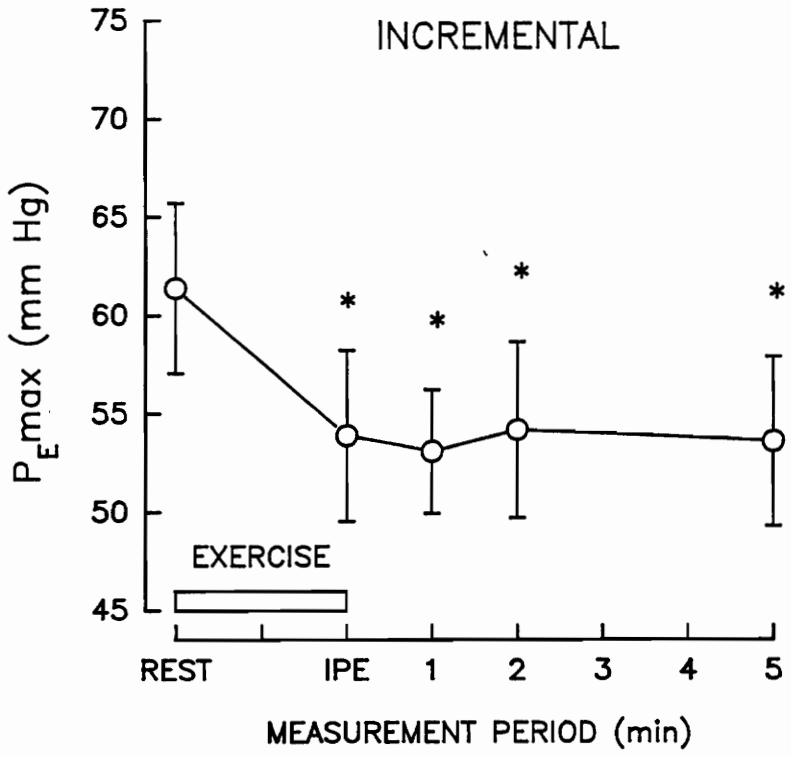
INCREMENTAL
75
70
65
60
55
50
45
$P_E max$ (mm Hg)
EXERCISE
REST
IPE
1
2
3
4
5
MEASUREMENT PERIOD (min)
*
*
*
*

Figure 1

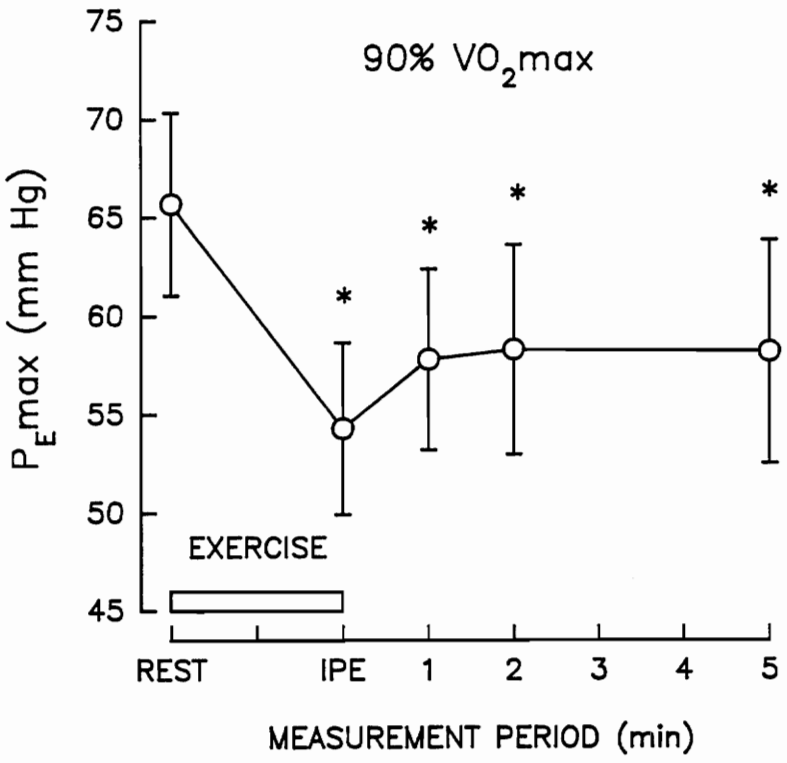
90% VO2max
PEmax (mm Hg)
75
70
65
60
55
50
45
EXERCISE
REST
IPE
1
2
3
4
5
MEASUREMENT PERIOD (min)
*
*
*
*

Figure 2

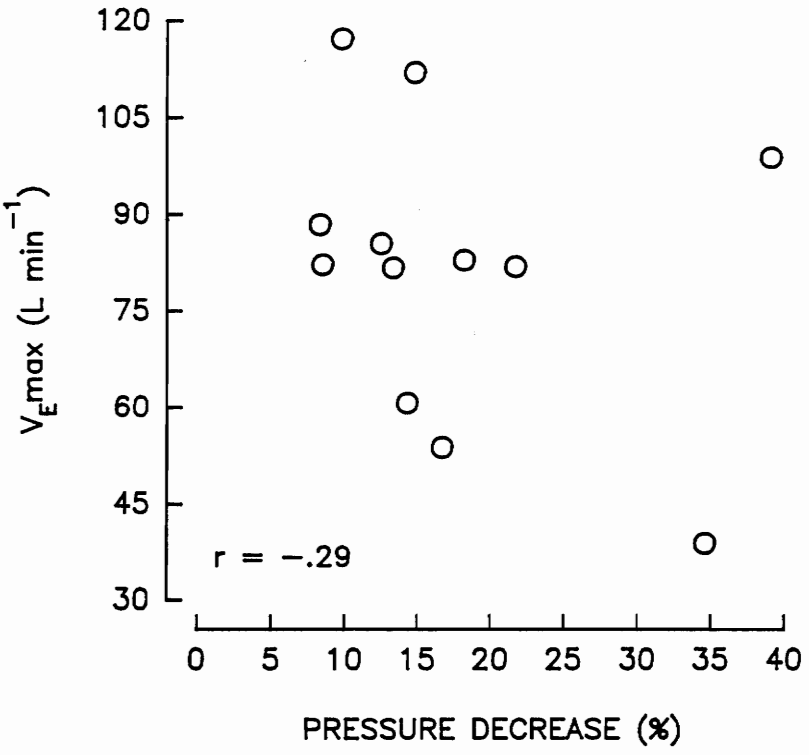
120
105
90
75
60
45
30
$V_E max$ (L min^{-1})
r = −.29
0
5
10
15
20
25
30
35
40
PRESSURE DECREASE (%)

Figure 3

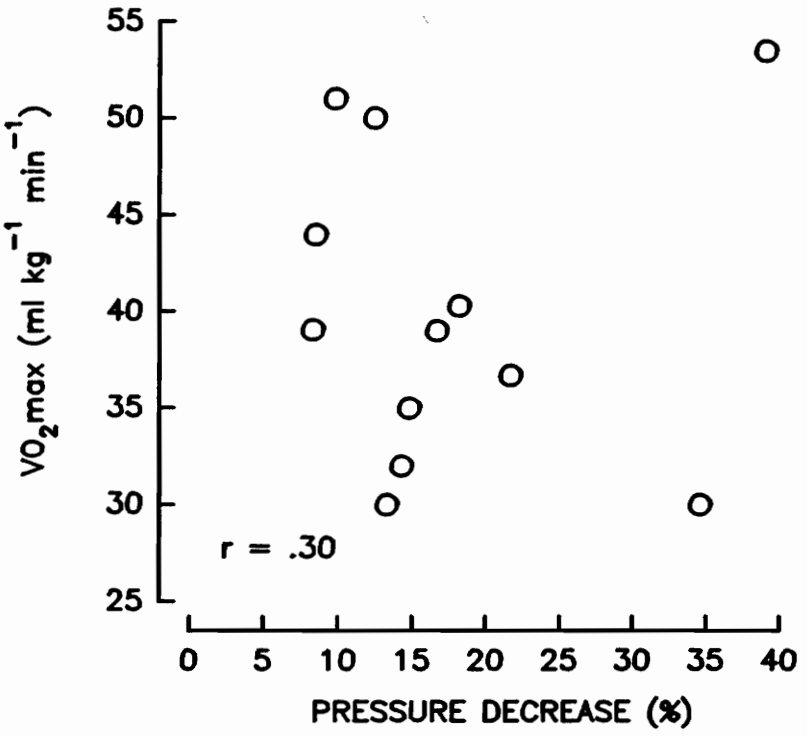
55
50
45
40
35
30
25
VO_2max (ml kg^{-1} min^{-1})
r = .30
0
5
10
15
20
25
30
35
40
PRESSURE DECREASE (%)

Figure 4

Chapter IV

SUMMARY

Recent literature evaluating maximal and short-term exercise performance has implicated expiratory muscle fatigue as a possible source of limitation at high exercise intensities (O'Kroy et al., 1991). The present investigation evaluated PeMax following two different short-term, intensive exercise bouts to determine whether this type of exercise stimulus provides the ventilitory demands great enough to result in a decline in expiratory muscle performance.

Twelve apparently healthy subjects not currently in active physical training underwent two different exercise protocols; a graded exercise test on a cycle ergometer and an exercise bout to exhaustion at a workload corresponding to 90% of VO_2 max. PeMax was measured before the exercise trials as well as immediate post, one, two and five minutes post exercise.

Subjects cycled for a mean duration of 13 min on the graded protocol to a mean VO_2 max of 40 $\pm$ 2.3 ml/kg/min with a coefficient of variation of .58. PeMax was found to be significantly lower immediatly post exercise, (53.9 $\pm$ 4.3 mmHg), as compared to resting values (61.4 $\pm$ 4.3 mmHg). This decline in PeMax persisted throughout the five minute recovery with PeMax values of 53.1 $\pm$ 3.1 mmHg, 54.2 $\pm$ 4.5

mmHg, and 53.6 ± 4.3 mmHg at one, two and five minutes respectively.

PeMax was also found to decline after the constant workload, high intensity bout. Subjects cycled for a mean duration of 5.5 min. at a workload corresponding to 90% of their predetermined VO_2 max. PeMax dropped from a mean resting value of 65.75 ± 4.7 mmHg to 54.28 ± 4.4 mmHg immediately post exercise. This decline in PeMax persisted throughout the five minute recovery with values of 57.84 ± 4.6 mmHg, 58.29 ± 5.3 mmHg, and 58.16 ± 5.7 mmHg at one, two and five minutes respectively.

No significant difference in the pattern of PeMax change was found between the two trials. In addition, no significant relationship was found between the decline in PeMax and VO_2 max or decline in PeMax and Ve max.

DISCUSSION

The present study demonstrated that both the graded maximal exercise and constant workload high intensity exercise resulted in a decline in expiratory muscle performance. This suggests that these two exercise protocols caused a ventilitory stimulus great enough to affect expiratory muscle function in apparently healthy college aged individuals not in active physical training. This finding is in agreement with

other research in this area reporting declines in both inspiratory and expiratory muscle function in response to intense exercise.

Studies evaluating expiratory muscle performance suggest that the expiratory muscles are recruited in response to increased ventilitory demand (Loke et al., 1982). Research demonstrates that although the muscles of expiration do not contribute to the work of passive breathing, these muscles are recruited at ventilations of 40 L/min (Campbell and Green, 1955; Strohl et al, 1981). The present study found a mean maximal ventilation of 82 $\pm$ 6.5 L/min. This would indicate that prolonged activity at moderate to high ventilations may be capable of causing expiratory fatigue. This is supported by studies evaluating marathon runners after a race, which report declines in PeMax of 27.9% as well as reductions in FVC (Loke et al., 1982).

The present investigation supports the idea that expiratory muscle dysfunction may occur in response to short, intensive exercise. Although very few investigations have examined expiratory muscle performance using this type of exercise stimulus, research in this area support the possibility that it may occur. Declines in PeMax have been reported in response to a seven minute bout at 90% of VO_2 max and significant declines in FVC have also been reported using this protocol (O'Kroy et al., 1991). The present study found

a decline in PeMax following both maximal graded exercise and a constant workload bout at 90% of VO_2 max which supports the findings of O'Kroy et al. (1991).

The magnitude of decline in PeMax found in this study is in agreement with declines seen in studies evaluating inspiratory muscle performance. Although patterns of recruitment are different between these two muscle groups, it is relevant to compare them because together they share the increased work of breathing associated with high ventilations. Bye et al., (1984) reported a 28% decline in Pdi as well as a 20% decline in the ratio of high to low frequency components of the diaphragm EMG after short term high intensity constant workload exercise bout. In addition, Coast et al., (1990) reported a 17% decline in PiMax in untrained subjects after maximal graded exercise. These studies are in agreement with the mean 14.5% decline in PeMax found in the present study. These investigations suggest that both inspiratory and expiratory muscle performance declines in response to short-term, high-intensity exercise, and the magnitude of decline is similar between the two muscle groups.

The possibility exists that the declines in PeMax found in this study may be attributed to a lack of motivation to perform maximal breathing maneuvers after intensive exercise. If this were the situation, the declines in PeMax would reflect a generalized fatigue associated with the discomfort

of exercise rather than fatigue of the expiratory muscles. This is unlikely due to several reasons. First, as previoulsy mentioned, the magnitude of decline in PeMax in this study is consistent with other studies evaluating respiratory muscle function in response to short-term, high-intensity exercise. Second, the magnitude of decline in PeMax was not significantly different between the two trials in this study. It is unlikely that a lack of motivation would be so consistent in response to two different trials that were of different duration on separate days. Finally, declines in PeMax persisted throughout the five minute recovery for both exercise trials in this study. If the discomfort associated with high intensity exercise resulted in a a lack of motivation to exhale maximally, it would be expected that PeMax would return to resting values once the subject had a chance to recover from the discomfort of exercise. Since PeMax values were still depressed five minutes into recovery, factors other than motivation were causing the decline. Thus, the declines in PeMax seen in this investigation represent expiratory muscle dysfunction rather than a lack of motivation to perform maximally in these particular subjects.

The decline in PeMax throughout recovery seen in this investigation is consistant with other studies in this area. The declines in PiMax found by Coast et al. (1990), after maximal graded exercise persisted throughout a five minute

recovery. These findings are in agreement with O'Kroy et al. (1991), who reported declines in PeMax five minutes after a seven minute bout at 90% of VO_2 max and a 30 min bout at 60% of VO_2 max. Additionally, Suzuki et al., (1991) reported a decline in PeMax for up to 60 min following expiratory resistive loaded breathing but did not find a corresponding EMG pattern of fatigue. Although the machanism behind this decline in PeMax is not known, it is possible that the declines in expiratory muscle performance may be persistant enough to affect subsequent exercise performance.

The present study did not find a strong correlation between the magnitude of decline in PeMax and VO_2 max. This conflicts with research which found that highly trained subjects have lesser changes in PiMax following maximal exercise than untrained subjects (Coast et al., 1990). The different changes in PiMax betweem trained and untrained subjects reported in this study were attributed to a possible training of the respiratory muscles. One reason for a failure to see a relationship between PeMax and VO_2 max, as well as a relationship between PeMax and Ve max in this study could be due to the homogeneous population of untrained subjects. This present study consisted of subjects with a mean VO_2 max of 40 ml/kg/min $\pm$ 2.3 with a coefficient of variation of .58. In addition, none of the subjects in the present study would be considered to be trained and thus may not consistently reach

ventilations high enough in their daily activities to result in a training effect of the respiratory muscles.

RESEARCH IMPLICATIONS

The primary objective of this study was to evaluate expiratory muscle performance during two short-term, high-intensity exercise protocols. The declines in PeMax observed in response to both graded and constant workload maximal exercise suggest that this type of exercise stimulus provides the ventilitory demands great enough to cause a decline in expiratory muscle performance.

This finding provides a framework for further research in order to determine if expiratory muscle dysfunction is ultimatly a limiting factor on exercise performance. This could have implications not only for the athlete, training to optimize exercise performance, but also in the diagnostic and functional assessment of VO_2 max of sedentary populations. Since greater declines in respiratory muscle performance are seen in sedentary people, respiratory system limitations may occur in this population that currently have been attributed to limitations in Vo2 max. In addition, recent research in the area of respiratory muscle function suggests that a training effect on the muscles of respiration may be possible in response to repeated exposure to increased ventilations. If this were to be confirmed, athletes may alter their training

to optimize these potential benefits. This also may have implications for exercise training in patients with COPD and efforts to improve their ability to perform activities of daily living.

RECOMMENDATIONS FOR FURTHER RESEARCH

In order to fully understand the significance of the decline in PeMax found in this investigation, further research is necessary. The following are suggestions for futher research relating to expiratory muscle performance, as well as subsequent exercise performance.

1) Since research indicates that PiMax declines to a greater extent in trained subjects rather than untrained subjects, it would be beneficial to repeat the present investigation using highly trained athletes. By comparing the magnitude of decline found in trained and untrained subjects, more information could be gathered regarding the possibility of a training effect of the expiratory muscles.

2) Another important research question in the area of expiratory muscle function is whether the declines seen in PeMax have an effect on exercise performance. The answer to this question could help to determine the importance of further study in the area of expiratory muscle fatigue. Therefore, it would be beneficial to evaluate exercise performance after inducing a decline in PeMax perhaps by

external loaded breathing. A decline in subsequent exercise performance has been demonstrated after the induction of inspiratory muscle fatigue, which suggests that it may occur in the expiratory muscles.

3) Another important area of investigation in order to determine the magnitude of expiratory muscle dysfunction is to determine the point at which expiratory muscle performance begins to decline. Therefore, it would be valuable to repeat the present investigation using an intense, but not exhaustive protocol in order to determine if this stimulus would be sufficient to cause a decline in PeMax.

4) Finally, it is important to investigate possible mechanisms behind the decline in expiratory muscle performance. Although it is postulated that the declines seen in PeMax are a result of an increased work of breathing associated with exercise, this has yet to be investigated fully. Therefore, in order to confirm whether the decline in PeMax during intensive exercise is due to an increased mechanical work of breathing, studies utilizing a lower density gas would be beneficial. A lower density gas such as helium would decrease the work required by the expiratory muscles. This would allow researchers to examine the effects of a decreased workload on PeMax at exercise.

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Appendix A

METHODOLOGY

METHODOLOGY

Subjects

Apparently healthy college aged male (n=7), and female (n=5) volunteers were used for this investigation. In order to qualify for participation in this study, individuals had to be nonsmokers, have no history of chronic pulmonary disease and have had no recent upper respiratory infections. In addition, only individuals who had not participated in any regular aerobic training for three months prior to the investigation were selected to participate. Subjects were screened by completing the questionnaire presented in Appendix C. FEV_1 values were measured both before and after exercise to eliminate any subjects with present pulmonary dysfunction.

General Protocol

Subjects performed a graded exercise test on a cycle ergometer in order to determine VO_2 max, and returned on a second day for a short-term, high-intensity cycling bout at a workload corresponding to 90% of VO_2 max. Each test required subjects to exercise to voluntary exhaustion. Measurements of PeMax were obtained before each trial, immediate post-exercise and during recovery. Trials were separated by two to ten days.

Measurement of PeMax

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

Subjects, wearing a noseclip, were first directed to inhale to total lung capacity. The "airway" was closed and each subject was instructed to exhale maximally against the resistance tube. A minimum of three resting trials were obtained before each exercise bout. More than three trials were performed if significant variation existed between the second and third trials. Measurements were also taken after both exercise trials, including immediate post (taken within twenty seconds of test termination), one, two and five minute recovery measurements. Subjects were verbally encouraged to give a maximal effort during each trial. Pilot data revealed a high reproducability between trials.

Measurement of $FEV_{1.0}$

Forced expiratory volume during the first second of exhalation was measured both before and five to seven minutes after the graded exercise trial. Subjects were directed to inhale to total lung capacity and then exhale 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_2 max test.

Graded Exercise Test

For the graded exercise test, males started at a workload of 1.0 kg, and females at a workload of 0.5 kg at a constant rate of 50 revolutions per minute. Workload was increased 0.5 kg every two minutes until the subject was unable to continue despite verbal encouragement. During the final 30 sec. of each stage, heart rate, blood pressure, perceived exertion, ventilation and expired gas fractions were determined. Measurement of VO_2, VCO_2, and Ve were made by the open circuit technique 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_2 max included a respiratory exchange ratio greater than 1.15, a heart rate $\pm$ 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 were met, then the highest VO_2 recorded was chosen as the subject's VO_2 max. All subjects successfully met this criteria. PeMax measurements were taken both before and after the test by the technique described previously.

Constant Workload Exercise

Subjects reported to the lab on a second day in order to perform a short-term, high-intensity, constant workload exercise bout. After a two minute warm up period, subjects cycled at a workload corresponding to 90% of their VO_2 max. They cycled at this workload until they were unable to continue despite verbal encouragement. During this exercise trial, heart rate, ventilation, and expired gas fractions were determined. PeMax measurements were performed as described previously.

Statistics

To determine the effects of exercise protocol and measurement period on PeMax, a two-way ANOVA, adjusted for repeated measures was performed. Where indicated, a Duncan's post-hoc exam was used to determine individual differences.

Appendix B

INFORMED CONSENT

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.

2. Risks and Discomforts

There exists the possibility of certain changes occurring during the test. These include abnormal blood pressure, fainting, disorder of 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 importnace. 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 the diagnosis of an illness or in evaluating what type of physical activities you might do with low risk of harm.

5. Inquiries

Any questions about the procedures used in the exercise test or in the estimation of functional capacity are encouraged. 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

Appendix C

QUESTIONNAIRE

Name _____ Date _____

Age _____

Subject number _____

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. Are you currently participating in any regular aerobic exercise? If yes, please indicate type of exercise and frequency.

Appendix D

RESULTS OF REPEATED MEASURES ANOVA AND POST HOC TESTS

Table 1

Mean and Standard Error for Age, Height, Body Weight, VO_2 max, Ve max, and $FEV_{1.0}$.

Variable	Mean	S.E.
Age (yrs)	20.5	0.23
Height (cm)	168.46	2.50
Weight (kg)	67.13	2.42
VO_2 max (ml/kg/min)	40.05	2.34
Ve max (L/min)	82.04	6.50
$FEV_{1.0}$ (L)	3.42	0.11

Table 2

Analysis of Variance Between Measurements Periods for the 12 Subjects during both the Graded and 90% VO_2 max Trials.

Source	DF	Sum of Squares	Mean Square	F Ratio	Prob>F
Test	13	93.50	393.50	0.36	NS
Subj(A)	22	24251.52	1102.34		
Time	4	1341.77	335.44	9.31	P<.05
Test(B)	4	79.91	19.98	0.55	NS
Error	88	3169.01	36.01		
Total	119	29235.71			

A=Test
B=Time

Table 3

Duncan's Post-Hoc Analyses of Graded and 90% VO_2 max Trials.

Graded Exercise		90% VO_2 max	
Time	Pressure (mm Hg)	Time	Pressure (mm Hg)
Rest	61.4 ± 4.3	Rest	65.7 ± 4.7
I-Post	53.9 ± 4.3 *	I-Post	54.3 ± 4.4 *
1-Post	53.1 ± 3.1 *	1-Post	57.8 ± 4.6 *
2-Post	54.2 ± 4.5 *	2-Post	58.3 ± 5.3 *
5-Post	53.6 ± 4.3 *	5-Post	59.7 ± 5.7 *

Appendix E

RAW DATA

Table 3

RAW DATA

SUBJECT	TEST	REST	IPE	POST1	POST2	POST5
1	1	77.5	50	63.75	53.75	53.75
1	2	79.4	67.65	67.65	64.71	70.59
2	1	71.88	79.69	51.56	54.69	65.63
2	2	88.24	73.53	67.65	67.65	79.41
3	1	67.39	45.31	54.69	48.44	48.44
3	2	71.88	43.75	59.38	59.38	60.16
4	1	57.81	48.44	50.78	53.13	53.13
4	2	56.25	51.56	49.22	52.34	49.22
5	1	39.38	36.88	40	38.75	37.5
5	2	51.47	44.12	44.12	47.06	38.24
6	1	32.81	23.44	27.34	29.69	28.13
6	2	40.63	26.56	25.78	26.56	25.78
7	1	63.23	70.59	61.76	58.82	61.76
7	2	66.18	57.35	57.35	61.76	61.76
8	1	62.5	62.5	60.9	65.23	60.9
8	2	66.25	51.88	60	63.13	62.5
9	1	63.75	53.75	53.13	60	55
9	2	63.75	57.5	75	60	60
10	1	86.76	63.24	66.18	92.65	86.76
10	2	94.12	82.35	86.76	101.47	97.06
11	1	51.47	52.94	50	50	47.06
11	2	46.09	42.19	45.31	35.16	34.38
12	1	62.5	60	57.5	45	45
12	2	64.71	52.94	55.88	60.29	58.82

VITA

Lisa Jacqueline Wilkins was born July, 2, 1968 in Denville, New Jersey to Mrs. Marjorie Wilkins and Mr.Douglas Wilkins. She has one older sister, Debbie, who is currently teaching middle school in Hillsborough, New Jersey, and one younger brother, Gordon, who will graduate from Washington and Lee University in Lexington, Virginia, this June. Lisa graduated from Mountain Lakes High School in 1986, and from there went on to attend Furman University in Greenville, South Carolina. At Furman, Lisa majored in biology, but became interested in exercise science and took several supporting courses in this area. She obtained a B.S. degree in Biology from Furman in 1990, and began courses toward her Master of Science Degree in exercise physiology at Virginia Polytechnic Institute and State University in Blacksburg, Virginia later that same year. While a graduate student, Lisa worked in the Virginia Tech Cardiac Rehabilitation Program. In addition, Lisa worked in the Cardio-Pulmonary Department at Montgomery Regional Hospital as a cardiac rehabilitation educator, assisting in exercise testing, phase I, and phase II cardiac rehabilitation. After obtaining her masters degreee, Lisa would like to work in a hospital based cardiac rehabilitation program.

Lisa Jacqueline Wilkins