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by

Terry Griffin

THESIS

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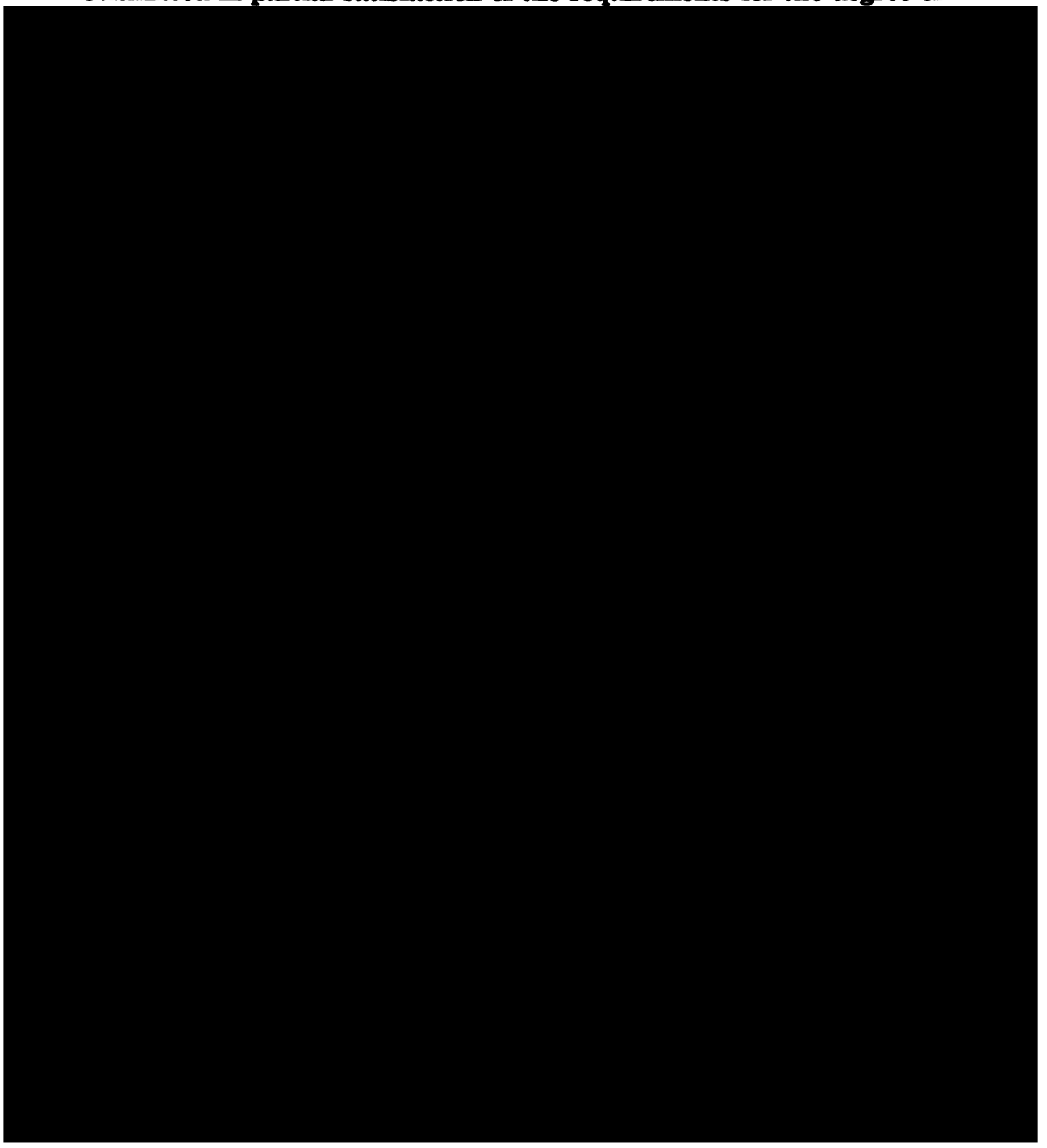

University of California, San Francisco

Department of Physiological Nursing

School of Nursing

THESIS:

Are Changes In Dyspnea Related To Changes In Exercise Capacity After Twelve Exercise Sessions In Chronic Obstructive Pulmonary Disease Patients?

Terry Griffin, RN, MS Candidate

June 16, 1994

Dedication

Thoughout the long and late hours of study
I was never without the warm and embracing thoughts of *my boys*;

I dedicate this thesis to
my loving and supportive husband, Kevin
&
to my very special sons, Jack and Ben

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Tables:

People with chronic obstructive pulmonary disease (COPD) have decreased activity tolerance due to the subjective sensation of dyspnea as well as limited lung function, cardiovascular and muscular strength and endurance. COPD patients with moderate or severe airflow obstruction are limited primarily in their exercise tolerance and activities of daily living by ventilatory capacity or limits in pulmonary gas exchange, unlike sedentary normals, who are limited by hemodynamic capacity (Casaburi,1993). Symptoms of dyspnea and decreased activity tolerance lead progressively to a state of deconditioning and further activity intolerance, which foster a subsequent, worsening debilitative state. This chronic debilitative state places patients at risk for increased exacerbations of the disease and subsequent hospitalizations, as well as loss of productive days at work, independence with activities of daily living and consequently, higher rates of depression and lower self esteem (Gift & Cahill, 1990; Gift, Moore, & Soeken, 1992; Light, Merrill, Despars, Gordon, & Multipassi, 1985). Since COPD is a chronic, progressive disease with no definitive cure, management of the illness focuses on improving functional status and preventing acute exacerbations, in hopes of slowing progressive debilitation.

In order to improve functional ability, therapies should include management of the symptom of dyspnea and prevention of progressive deconditioning. One management strategy that has been effective in enhancing activity tolerance and decreasing dyspnea in COPD patients is exercise therapy. Pulmonary rehabilitation programs emphasize exercise as a component of their programs, using strategies such as diaphramatic and pursed-lip breathing, and stress management techniques, to help minimize dyspnea during exercise. It is generalized that whatever changes in physical capacity improve might also be relatived to dyspnea. Symptom limitations to exercise are most often due to dyspnea in the COPD patient (Killian, 1993). If the sensation of dyspnea for given levels of activity or exercise can be diminished, COPD patients may be able to participate in daily activities more fully, and therefore, interrupt the progressive debilitative state cycle, allowing them to be more functionally independent. Therefore, the purpose of this study is to compare

exercise capacity to the change in dyspnea using the VAS scale, before and after a twelve session out patient exercise program for moderate to severe COPD patients.

Background

Although exercise therapy in the COPD patient population has been studied extensively over the past two decades, current trends and health care strategies emphasizing preventive care for chronically ill patients makes research in this area even more timely (DHHS, 1992). The purpose of exercise training in healthy subjects is to stress the body so that physiological adaptations that help the body tolerate the metabolic demands of exercise take place, improving exercise capacity (Brooks, 1985). A major purpose of exercise in pulmonary rehabilitation programs is to increase the efficiency of muscles to use oxygen (O2), and if intensity of exercise is high enough, to decrease the work of the lungs by decreasing ventilatory requirements through improved aerobic capacity thereby decreasing dyspnea (Casaburi, Storer, & Wasserman, 1987).

Mechanisms of Dyspnea - Limitation to Exercise

Dyspnea, the subjective sensation of difficult, uncomfortable breathing includes both the perception of labored breathing by the patient and the reaction to that sensation (Comroe, 1965). Both the sensory and reactive components, like the mechanisms of pain, are subjective. Mechanisms of dyspnea related to COPD are multifactorial, including both psychological and physiological factors (Carrieri-Kohlman, Janson-Bjerklie, & Jacobs, 1984). Studies about the phenomenon have led to the inclusion of several possible mechanisms, that contribute to individual perceptions and intensities of dyspnea unrelated to severity of disease (Killian, 1993).

Other than improvements in exercise capacity or increases in efficiencies of walking on the treadmill, another mechanism that may help improve the perception of dyspnea with exercise is desensitization. This concept related to COPD and exercise has been referred to frequently over the past few decades (Belman, 1991; Carrieri, Douglas Gormley & Stulbarg, 1993; Casaburi, 1991). Desensitization is the process by which

repeated exposure to a fearful stimulus, e.g., snakes, heights, or in this circumstance, dyspnea, gradually decreases anxiety or fear associated with the stimulus, making it more tolerable (Carrieri, Douglas, Gormley, & Stulbarg, 1993). Desensitization has been described as decreased dyspnea out of proportion to changes in ventilation (Carrieri et al., unpublished).. Belman et al., (1991) studied COPD patient in an exercise program and found that dyspnea scoring using a Borg rating scale decreased successively over the ten days, while the physiological measures of VO2, VE, HR, and RR stabilized over one or two attempts. This suggests that desensitization to dyspnea may have played a part in the improvement in the patients after exercise.

Recent study of the mechanisms of dyspnea suggests that dyspnea arises from central perception of afferent signals from chemoreceptors, lungs receptors and possibly mechanical receptors, and is altered by information from muscle receptors and central nervous system processing affected by emotional or psychological states (Stulbarg, 1994). Because COPD patients are limited by ventilatory and exercise capacity, response to exercise is different for them when compared to healthy subjects. A brief explanation of the physiologic changes that normally occur with exercise in healthy people will precede a description of those changes occurring in the COPD patient.

Response to Exercise in the Healthy Person

The function of the myocardium during exercise is to deliver oxygenated blood to the exercising muscle and essential organs by raising cardiac output (Oka, 1990). For the healthy person, with the increase in metabolic demand of exercise, pulmonary respiration and cardiac output will balance cellular respiration until maximum exercise is reached. Endurance exercise in the healthy person has structural, cardiovascular and psychological benefits. Endurance training increases maximal oxygen consumption (VO2), both because arterial-venous O2 content difference (A-VO2)widens and maximal cardiac output is higher (Pollock & Wilmore, 1990). Stroke volume is one of the most important determinations of individual variability in maximum oxygen consumption. The increase in

stroke volume during submaximum exercise is usually due to a redistribution of blood volume, which increases venous return. Both a decrease in heart rate and an increase in venous return enhance preload, which ultimately increases cardiac output (Oka, 1990).

Another key change after exercise in the conditioned healthy person is the skeletal muscle's shift in fuel usage from glycogen to more efficient fatty acids for oxidation, thereby allowing exercise to continue for a longer time, indicating increased exercise capacity (Oka, 1990; Casaburi, 1992). Oxygen extraction by the trained muscle is more efficient, which accounts for the widened A-VO2 at a given level of exercise (Casaburi, 1992; Hollosky & Coyle, 1984). In the healthy person exercise capacity is limited by hemodynamic factors, especially the level to which the cardiac output can be elevated efficiently and by the ability of the skeletal muscles to extract sufficient oxygen, so they do not become fatigued (Ries, 1991). No limitations are imposed by ventilatory capacity or pulmonary gas exchange, although the ventilation requirement may decrease (Casaburi, 1987).

At low-level exercise, cardiac output increases (primarily due to an increase in heart rate), A-V02 widens, and oxygen consumption (VO2) and carbon dioxide production (VCO2) are increased (Ries, 1991). As the exercise intensity is increased, cardiac output becomes inadequate to provide enough blood flow and oxygen to the exercising muscles at which time aerobic metabolism converts to anaerobic metabolism, known as anaerobic threshold (AT) (Ries, 1991). Lactic acid is produced during anaerobic metabolism, which leads to the accumulation of additional CO2. At this point, minute ventilation (VE) rises nonlinearly to the VO2, which indicates AT has been obtained (Wasserman, Hansen, Sue, & Whipp, 1987). Ventilation is stimulated both to clear the added CO2 load and because the metabolic acidosis directly stimulates carotid bodies (Wasserman, Whip, Koyal, & Cleary, 1975). Exercise capacity in the healthy individual is therefore limited by cardiac output and the resultant blood flow to the working muscles (Ries, 1991). Increased exercise capacity may be due to a "training

effect" whereby VO2 max increases, relative to the intensity of the training and the initial state of fitness. Enhanced exercise capacity may also be due to improved technique of performance of a given task rather than a conditioning effect. In this situation a decrease in VO2 for a comparable work load would be seen. Motivation is also recognized as a significant factor that may affect exercise performance both in training and in testing, and influence the change in aquired exercise capacity (Belman, 1986).

Exercise Limitation in the COPD patient

Although a standard sequence of exercise responses has been clearly described in healthy subjects, these classic physiologic principles are not possible during exercise in the COPD patient. Because these patients are limited by their ventilatory capacity, or by pulmonary gas exchange inefficiencies, patients with chronic obstructive pulmonary disease are primarily limited by dyspnea rather than hemodynamic factors (Ries, 1991). As the disease progresses, it takes less activity to foster more dyspnea and the COPD patient becomes fearful of engaging in activities that produce dyspnea. This leads the patient to avoid physical activity associated with these unpleasant symptoms leading to a fear-dyspnea inactivity cycle (Dudley, 1980). The patient becomes less active and generally more deconditioned with less exercise tolerance (Ries, 1991).

A number of factors that affect ventilation interact to reduce exercise tolerance and increase breathlessness during exercise in the COPD patient. COPD patients have decreased exercise capacity due to mechanical, cardiovascular, and physiologic factors of increased ventilatory requirement and decreased ventilatory capacity (Wasserman, 1993), as well as psychologic factors including the fear and anxiety associated with dyspnea (Gift & Cahill, 1990; Carrieri, Douglas, Gormley, & Stulbarg, 1993). Increased ventilatory requirements due to ventilation-perfusion (V□/Q) mismatching can lead to increased sensations of dyspnea and ultimately decrease activity tolerance in COPD patients, in contrast to healthy subjects who have normal V/Q in the lungs and are able to meet ventilatory demands of exercise. Patients with COPD have increased dead space

(VD/DT) due to V/Q mismatching. The increased VD/VT requires that VE increase more to blow off CO2 produced at the tissue level than is required in healthy subjects who have increased ventilatory demands with exercise (Wasserman, 1993). Arterial hypoxemia caused by the increased perfusion of poorly ventilated lung units may be another mechanism causing dyspnea. Resection of the carotid bodies which removes afferent signals sensative to the level of partial pressure of oxygen in the blood (PaO2), relieved dyspnea in a significant number of patients (Vermeire, 1987; Stulbarg, 1989). Development of lactic acidosis at low levels of exercise, due to their lack of conditioning, and increases in metabolic cost of work due to poor elastic recoil of the lungs are also factors associated with increases in ventilatory requirement and dyspnea in COPD patients (Wasserman, 1993).

Decreased ventilatory capacity due to airflow obstruction and decreased elastic recoil adds exercise limitation due to dyspnea in these patients. The hyperinflated state is a mechanical disadvantage and may add to the sensation of dyspnea because of the receptors in the intercostals which are sensitive to length-tension changes (Campbell, Agostoni, & Newsom-Davis, 1970). Dyspnea is associated with the development of respiratory muscle force in relationship to muscle fiber length, the frequency of motor unit action potentials, and the velocity of muscle contraction (Killian, Summers, Basalygo, & Campbell, 1985). O'Donnell & Webb (1992), studied breathlessness in 37 patients with chronic airflow obstruction. They found that those with a higher baseline dyspnea index (BDI) score had different pathophysiologic responses to exercise than those who had significantly ($p < 0.001$) lower BDI scores. Severely breathless patients had lower resting diffusing capacities and accelerated ventilatory responses to exercise. Ventilatory responses for a given metabolic load were, on average, 33 percent higher ($p < 0.05$) in the severely breathless group reflecting greater ventilation-perfusion mismatch and wasted ventilation. In a more recent study (Webb, Bertley, McGuire, Samis & O'Donnell, 1994), it was found that after a six week exercise program decreases in dyspnea were related to a

fall in ventilatory demands (ΔVE/VO2) during exercise, as well as desensitization to muscular effort. Dynamic hyperinflation which imposes elastic and inspiratory threshold loads in in obstructive disease also had recently been noted to influence dyspnea (Loughseed,Webb, & O'Donnell, 1994).

Depending on severity of disease, these patients may also be limited in their exercise capacity by impaired cardiac output. Stroke volume is lower in COPD patients, decreasing their cardiac output response to exercise. This lower stroke volume is due to decreases in preload because of high intrathoracic pressures throughout the respiratory cycle coupled with elevated pulmonary vascular resistance (Chester et al., 1977). Preload may decrease further with exercise as breathing rate increases. An indirect noninvasive measure of stroke volume is oxygen pulse, oxygen uptake per heart beat, (VO2/HR). This measurement is low in COPD patients due to decreased stroke volume. Although their oxygen pulse VO2/HR is low, it continues to increase during exercise, but at a lower slope than in normals (Nery, Wasserman, & French, 1983).

Over the past few decades, studies that have investigated COPD patients' response to exercise programs have found that depending on intensity, duration and frequency of exercise and length of the program, walking endurance and dyspnea improved. Exercise capacity variables also changed in response to exercise (Punzal et al., 1991; Casaburi et al., 1991). Based on the physiologic mechanisms of improvements in exercise capacity in normals, it would be expected that the submaximum physiologic variables: heart rate (HR), respiratory rate (RR), minute ventilation (VE), would decrease and that oxygen pulse (VO2/HR) would increase at similar workloads (VO2). If VO2 decreased it would be due to increased technique of performance of a task, rather than a conditioning effect (Belman, 1986).

Literature Review

Exercise and Dyspnea: Multidimensional Pulmonary Rehabilitation Programs

Neiderman (1980), studied 33 COPD patients, mean age 65, with mean FEV1

1.2L ± 0.8L. FEV1% predicted was not given to standardize severity of the patients, but it was noted that 17 of the 33 who completed the study had an FEV1 <1L, therefore at least half had severe obstructive disease. In this uncontrolled study, the subjects participated in a nine week pulmonary rehabilitation program, three times per week, for a total of 20 minutes of exercise a day. The exercise protocol included upper body exercises, free weights, and cycle ergometer, with initial intensity at 50% max. cycle ergometer wattage for 20 minutes or until symptom limited, with 25% increases in intensity every week. In each session, subjects were exercised to the same level of effort, as measured by the perceived exertion (RPE) scale. Maximal percent intensity was not reported by the investigator. After nine weeks, all subjects significantly increased their 12 minute walk time from a mean of 1349 ± 626 ft to 1700 ± 670 ft, ($p<0.01$), and maximal sustained workload measured as cycle time of 5.0 ± 0.9 to 12.4 ± 4.3 mins. The only significant physiologic change was a decrease in ventilatory equivalent for O2 (VE/VO2). There was no change in VO2 max., VE max., or O2 pulse at end exercise. Dyspnea significantly decreased, but did not correlate with changes in the twelve minute walk or maximum exertion. Mean change in dyspnea was not given and, therefore, the magnitude of change in dyspnea is difficult to evaluate. The most improvement in performance was found in those with the worst initial performance, which correlated with severity, measured by FEV1. There was no improvement in pulmonary function.

In a randomized controlled study Cockcroft, Saunders, & Berry (1981) investigated the effect of a standard graduated exercise program on pulmonary function, exercise capacity and dyspnea in 39 men with COPD (mean age 61.2 ± 5.02, mean FEV1 1.4L ± 0.57L). Because this study involved patients with only moderate obstructive disease its results may not be generalizable to more severly obstructed subjects. The treatment consisted of a 6 week supervised inpatient rehabilitaion program, followed by a ten week unsupervised home exercise program. The TG participated in an exercise program including cycle ergometry, rowing, swimming, and walking twice daily over

increasing distances of up to two miles. The intensity and duration of exercise was determined by the patients and recorded on a personal progress card. On discharge they were given verbal and written instructions to continue with simple exercises at home. The control group (CG) was given no exercise advice, but enterd the six-week exercise program after the treatment group (TG) group completed their four month exercise period.

There was no significant improvement in pulmonary function in either group. The 12 minute walking distance was the most sensitive index of differences in changes between the two groups after rehabilitation. After two months the distance increased a mean of 120 ±119 meters in the TG and 43±111 meters in the CG. The only physiological measure that differed significantly between the two groups was the maxVO2 ($p < 0.05$) which was measured on a treadmill; VO2 and VE did not decrease in response to similar work rates. By four months the difference between the two groups in the improvement in the 12 mw was no longer statistically significant. This may have been due to the gradual improvement in the CG in preparation for entry into the rehabilitation program after the four month CG period was completed. Fifty percent of the TG had improvement in their symptoms of dyspnea ($p<.01$) after two months, whereas only 6% of the CG had improvement after two months. A critique of the studies findings on changes in dyspnea is that there was no report of method to measure dyspnea. Subjects were asked if there dyspnea had improved or not. The study also did not indicate at what point in the study they were asked about their dyspnea. It was not indicated if the subjects were asked during exercise or retrospectively. Therefore reports of changes in dyspnea were not well contolled and the results may not be valid.

Strijbos, Koeter, & Meinesz (1990), studied the association of exercise and dyspnea in 30 COPD patients, average age 60 ± 4.8 years, mean FEV1 1.1L±0.48L, in a controlled correlational design. Two groups were randomly assigned to either a TG, who completed 24 thirty-minute sessions over twelve weeks during the pulmonary

rehabilitation program (PRP) or a CG, that received no pulmonary rehabilitation. Exercise testing was performed using incremental symptom-limited cycling. Dyspnea intensity was measured using the Borg scale. Mean maximal workload on the cycle ergometer and dyspnea scores decreased in the treatment group for the same workload level ($p< 0.05$) although intensity as a percent of heart rate or maxVO2 was not mentioned. No significant changes for dyspnea or workload occurred in the CG. While these investigators compared changes in exercise endurance and dyspnea in a controlled design, no measures of physiological change were reported to determine if htere was a conditioning effect of 12 weeks of exercise. Changes may have been due to becoming more mechanically efficient at walking or cycling, attention and positive encouragement received through the program, and/or desensitization.

One of the key variables in exercise programs in COPD patients is intensity of workload. Ries and Archibald (1987), found that many patients with severe lung disease can exercise at a high percentage of maximum exercise tolerance that is close to, or may exceed the highest level reached on the initial maximum exercise testing. Prediction equations have been developed for COPD patients to estimate VEmax based on the FEV1 (Spiro, 1977). It has been suggested that if COPD patients fail to exercise at the predicted VEmax, poor motivation may be a factor (Belman, 1986). Exercising at high intensities is based on the observation that ventilatory-limited patients can still sustain ventilation at a high percentage of their MVV (Casaburi et al., 1991).

Casaburi et al., (1991) examined the effect of reduced blood lactic acid levels on lowering the level of ventilation after high intensity aerobic training. Compared to previous studies these investigators studied a relatively younger group of COPD patients than previous studies (mean age 51± 9), who had less severe pulmonary function, FEV1 1.8 ±0.53L and FEV1% predicted 56 ±16%. Therefore, their findings may not be generalizable to COPD patients with severe or very severe airflow obstruction. The small group of 11 patients was randomly assigned to a high intensity (HI) work group and a low

intensity work group (LI). The subjects exercised for 45-120 minutes per day, 5 days a week for 8 weeks at 90 % of the anaerobic threshold (AT) or at a proportionally longer time at a lower work rate (60 % of the difference between AT and VO2 max) so that the same same total workload could be compared. The AT was determined from the plot of VCO2 versus VO2 during the initial incremental stress test. This noninvasive measure has been found to closely approximate the point at which blood bicarbonate begins to fall and the AT also falls (Patessio et al., 1993).

Significant reductions in lactate, both after high and low intensity exercising were noted, while VE max. significantly decreased only in the high intensity group. Endurance time increased by 73 % in the high intensity group, with a nonsignificant increase in the low intensity group. Although VEmax decreased, the subjective sensation of dyspnea was not measured.

Punzal & Ries (1991) reported the effect of exercising at high exercise intensities close to and above AT with only their exercise group from a larger study. The 52 subjects in this study had an mean FEV1 of 1.38± 0.67L, and mean age of 61.6 ± 8.3 years. The treatment group (n=18) exercised two times per week for four weeks, and one time per week for another four weeks at workloads close to AT, as well as exercising daily at home and keeping journals. The control group (n=34) trained at exercise intensities which were determined during the initial incremental stress test to have no evidence of significant lactic acidosis at maximum exercise. AT was determined noninvasively by examining the pattern of changes in the ventilatory equivalents of O2 (VE/VO2) and CO2 (VE/VO2), as well as the respiratory quotient (R), with increasing workloads. The supervised incremental exercise program consisted of walking on the treadmill for 20 minutes at high intensities up to 85% of baseline maximum, or until tolerated. Both groups increased thier mean endurance to the same degree, as well as increasing their VO2 max., and decreasing their dyspnea. Dyspnea ratings (modified Borg scale), decreased progressively through the eight week sessions. Ratings of dyspnea decreased from 3.6 +/- 1.6 at week 1 to 3.1

+/- 1.7 at week 4 and 2.6 +/- 1.6 at week 8 ($p < 0.0001$ by repeated measures ANOVA). the reported study, like most studies up to this time did not control for the influence of the educational component of comprehensive pulmonary rehabiliation on exercise and dyspnea outcomes.

To control for the covariable of education in PRPs on both dyspnea and exercise capacity, a larger study completed recently by this same research group (Reis, Kaplan, Limerg & Prewitt, 1994) randomly assigned COPD subjects to either comprehensive pulmonary rehabilitation (n=57) or to an education control group (n=62) a prospective experimental design, which followed patients for six years after treatment. The pulmonary rehabilitation program consited of 12 four-hour sessions over 8 weeks including education, physical and respiratory care instruction, pyschosocial support, and supervised exercise training. The education group attended 4 two-hour sessions over eight weeks including video tapes lectures, and discussion of similar topics. Outcome measures included pulmonary function, maximum exercise tolerance, and endurance, gas exchange, symptoms, self efficacy, depression, general quality of well being, and health care utilization. In comparison to the education group, the pulmonary rehabilitation group showed significantly greater improvement in measures of exercise tolerance and endurance, symptoms of breathlessness and fatigue, and walking self-efficacy after six years. There were no differences in lung function, depression, or quality of well being. Differences between the groups diminished after one year of follow-up. This study found that exercise is a beneficial component in improving exercise tolerance and decreasing symptoms in COPD patients. This preliminary abstract did not indicate how significantly dyspnea and exercise performance improved in the treatment group compared to the control group. This later study adds greatly to the body of knowledge related to exercise and its effect on dyspnea and exercise tolerance in COPD patients.

Although COPD patients in these studies all had improvements in the sensation of dyspnea and endurance, and some had improvements in physiologic measurements of

aerobic capacity, most of these studies were part of multifaceted PRPs of which educational components may have played a key role in influencing the outcomes of exercise capacity and dyspnea. These studies point out that the longer the program, the more frequent and intense the sessions, the greater the improvement in exercise endurance and exercise capacity changes. Exercise programs in which subjects work at exercise loads close to or above their AT can improve physiologic parameters of exercise capacity.

Exercise and Dyspnea: Studies of Exercise only, without Pulmonary Rehabilitation

Two classic studies using only exercise as the treatment investigated the response to exercise in COPD patients not involved in a formal PRP. McGavin, Gupta, Lloyd, & McHardy (1977), studied 24 male chronic bronchitis patients, mean age 61, FEV1 1.0 L, in a randomized controlled study. The treatment group performed stair climbing 12 weeks, five times per week, five to ten minutes per day at home. The treatment group was seen in two weeks, and monthly thereafter, for a total training time of three months. The control group was sent home after initial testing without instructions to exercise. Both groups completed questionnaires that rated the sensation of dyspnea, well-being, and general activity, cough and sputum measures. The treatment group increased their walking distance by 6 %, whereas the control group decreased their distance by 2 % in a twelve minute walk. Statistical significance was not mentioned. Dyspnea significantly decreased in the treatment group ($p < .01$), but not in the control group. There was no significant improvement in heart rate, ventilation, or respiratory exchange ratio in either group during an incremental stress test.

Sinclair and Ingram (1980), studied 33 chronic bronchitis patients (FEV1 of 1.1 ± 0.5L, mean age 65.6 ± 8.5years), within an experimental design.The treatment group walked daily, stair climbed according to ability and symptom limitation, and completed home exercise diaries. The treatment group returned once a week for supervised exercise for twelve months. Exercise endurance on the treadmill improved significantly in the

treatment group, reaching a maximum plateau of a 24 % increase in walking distance after eight to twelve months. The treatment group also had a significant improvement in dyspnea ($p< 0.02$). Dyspnea was measured as deterioration, no change or improvement. Validity and reliability of the tool used was not reported. The control group had no significant improvements in distance walked in the 12 mw or in dyspnea. No significant improvements in HR, VE were noted in either group measured on the cycle ergometer. Muscular strength did not change significnatly in either group either, measured by a strain guage and muscle circumference.

Regardless of participation in pulmonary rehabilitation exercise COPD patients with wide ranges of obstruction improve their exercise endurance and report less dyspnea depending on intensities, duration of the program, frequency per week, initial level of conditioning and motivation. These improvements occur without appreciable changes in pulmonary function. Earlier studies (McGavin et al., 1977; Sinclair et al.; 1980, Neiderman, 1980; Cockcroft et al.; 1981, & Strijbos et al., 1990) exercised their patients at lower intensities below the AT, i.e., until symptom-limited, without appreciable improvements in aerobic capacity that might have induced a training effect measured by an increased VO2 max. These findings may be explained by many factors including the presenting deconditioned state, desensitization to dyspnea, increased motivation, and improved mechanical skill (Belman, 1986). Limitation to physiologic training effects in these studies may be due to the lack of sufficient training intensity (Casaburi et al., 1991). Later studies (Casaburi et al., 1991 & Punzal & Ries, 1991) have focused on exercising COPD patients at higher levels of intensity, close to anaerobic threshold. These investigators hypothesized that if lactic acid production could be reduced, the ventilatory requirement would be reduced, 0minimizing the work of breathing (Casaburi et al., 1991). If the ventilatory requirement is decreased, dyspnea should also decrease, and therefore exercise tolerance may improve. Although changes in exercise capacity and changes in dyspnea after varied exercise programs have been studied in COPD patients, the

relationship between these variables using valid and reliable tools has not been investigated.

Methods

Setting. Subjects were recruited from a large university medical center and its nearby community. Data was collected before and after twelve supervised treadmill exercise sessions. These exercise sessions were held three times a week, over a four week period in a large metropolitan teaching hospital in Northern California.

Sample. Fifty-two subjects (25 males, 27 females), ranging in age from 40 to 81 years, were recruited for the study. All subjects had less than 15% reversible airway disease after inhaling three puffs of albuterol (ATS, 1991) and had moderate to very severe obstructive pulmonary disease (FEV1 % predicted ranging from19 %- 61 % with a mean 38%, and a FEV1/FVC % ranging from 23 - 73%, with a mean of 42%) (Table 1). All subjects were evaluated for cardiopulmonary eligibility prior to the study by undergoing an incremental symptom-limited exercise stress test (Jones, 1982). Two subjects were on beta-blocking medication. Subjects were excluded from the study if they had active heart disease or other diseases that would interfere with the ability to exercise safely or if they had participated in a pulmonary rehabilitation program within the previous six months. Following an explanation of the possible risks and benefits of the study, informed consent was obtained from each subject and the original protocol was approved by the Committee on Human Research

Research Design

This correlational study is a secondary analysis of data from a larger study that examined the effect of nurse coaching on the components of dyspnea in exercising COPD patients. The subjects were randomly assigned to either a monitored exercise group (ME) or a nurse-coached exercise group (CE). The secondary analysis investigated the relationship between the change in noninvasive measures of exercise capacity and the change in dyspnea intensity (DI). There were two major analyses conducted. The first

analysis compared the variables at end exercise between the baseline incremental symptom-limited stress test 1 (ST1) and stress test 2 (ST2) following the twelve sessions of exercise. A second analysis compared the variables at the stage at end exercise at ST1 with the same variables at that same stage (Isostage) during stress test 2 (see Appendix for operational definitions). Once the subjects completed baseline spirometry a six 6 minute walk and physical exam, they returned within one week to perform the baseline incremental symptom-limited treadmill stress test (ST1).

Variables

The twelve individualized exercise session program was the independent variable or treatment. The dependent variables were exercise capacity variables and dyspnea intensity (DI) for a given level of work. The noninvasive physiologic variables measuring exercise capacity included oxygen pulse (VO2/HR), minute ventilation (VE), heart rate (HR), respiratory rate (RR), and peak achieved oxygen consumption (peak VO2) achieved during the stress test due to symptom limitation. Change in distance walked during the six minute walk and stages reached during the stress tests as measures of endurance also were measured. Every two minutes dyspnea intensity (DI) was measured using a vertical 200mm visual analogue scale (VAS), which has been shown to be a valid and reliable measure of perceived dyspnea in normal volunteers and COPD subjects (Adams, Chronos, Lane, & Guz, 1985; Gift, 1989). Subjects were asked "How short of breath are you?" and used a pencil to cross the solid line on the VAS scale for measurement of DI.

Original Study Procedures.

Incremental Symptom-limited Stress Test.

All subjects underwent an incremental symptom-limited stress test at baseline (ST1) and after the twelve exercise sessions (ST2). Subjects inhaled three doses of albuterol via MDI with an aerochamber under supervision. The exercise ST was performed within fifteen to sixty minutes of the first dose of albuterol.

Baseline physiological data was collected after consent was received. Resting

values for 12-lead ECG, oxygen saturation, (via finger probe Nellcor N-10), and metabolic cart (Quinton Q55 or Marquette) values were recorded, along with the cuff blood pressure, while walking on a Quinton Q50 treadmill (Quinton Instrument Co., Seattle WA). The treadmill procedure was explained and demonstrated to the subjects before they were tested. Demonstration emphasized proper technique of walking stride, posture and hand grip. Subjects were instructed to use two fingers on the treadmill handrail for balance only. Specific protocol hand signals were demonstrated to be used during the ST to communicate the subject's ability to continue with the stages of the test, or desire to stop because of limiting symptoms. Subjects were instructed that they should exercise to their maximum ability (peak exercise) and to walk until they could go no further.

The modified incremental exercise protocol (adapted from Punzal & Ries, 1991) included a three minute warm up at one mph and 0% grade was followed by incremental increases in speed of 0.5mph every 1.5 minutes, up to 3mph, with increasing grades of 2% every 1.5 minutes thereafter, up to a 16% grade. Subjects who were able to complete at least 3 minutes of warm up at one mph and one additional 90 second stage of exercise without signs of heart disease were invited to enter the study. Testing also included breath-by-breath analysis of expired gas, and continuous monitoring of pulse, 12-lead ECG, and oxygen saturation via finger probe (SpO2). Subjects were asked to rate dyspnea intensity every 1.5 minutes, 45 seconds into each successive stage. Blood pressure was recorded every two stages. For the purpose of this study, a stage is a 90 second interval at a predetermined speed and grade standardized in the protocol for the incremental stress test. The variables of interest for this secondary analysis included exercise capacity variables (VE, VO2, HR, VO2/HR, and RR) and dyspnea intensity (DI) compared at two separate workloads after the exercise treatment. The variables were compared to baseline end exercise values with stress test 2 at both end exercise and isostage. Isostage was the same stage reached during stress test 2 as achieved during baseline stress test1 at end exercise (Appendix).

The following criteria for stopping the maximal exercise test were used: 1) SpO2 < 80% for one full minute, 2) noncompensatory blood pressure changes, 3) arrhythmias, 4) ST wave changes, or 5) if symptom-limited maximum was reached (Jones, 1982). Symptom limitation for this sample included dyspnea, leg fatigue or generalized fatigue.

Standardized Protocol for Exercise Treatments - Independent Variable

The CE subjects were told that the purpose of the 12 exercise sessions was to make them short of breath in order to learn and practice different strategies to utilize while experiencing shortness of breath. The strategies demonstrated and provided by the nurse coach included: 1) short term goal setting by subject and nurse, 2) mastery of graduated subtasks whereby the nurse set small steps in the protocol to assure success, 3) modeling of effective coping strategies including: pursed-lip breathing, relaxation, and optimal breathing position/stride, 4) provision of protective aids and physical support by the nurse to reduce threat, 5) modulating severity of threat, 6) gradual extension of time coping with symptom, and 7) using social persuasion and emotion support (Williamson, 1985). Based on previous exercise session performances, the nurse encouraged subjects in the CE group to increase time, speed and/or grade on the treadmill each session and gave feedback and praise on their performance when the sessions were completed.

Subjects in the ME group were told that they could decide how long they walked, up to a 30 minute maximum, including 3 minutes each of warm-up and cool-down. They were not given encouragement. They were given feedback about HR, O2 saturation, treadmill time, speed, and grade during exercise only if requested by the subject. If subjects in this group requested more information about how to exercise optimally, they were referred by the nurse to the recommendations of the American Heart Association that exercise should be maintained for 20 minutes at 70% to 85% of their maximum heart rate.

The treadmill was calibrated prior to exercise treatment sessions. Subjects inhaled three puffs of albuterol before the treatment. All 49 subjects had a 3 minute warm-up at

0.7mph at all sessions. For the first treatment session (T1) of twelve sessions, stage 1 was programmed at 1 mph slower than maximum achieved in the ST with grade 0%. For the remaining sessions (T2-T12), subjects started at 2 stages below target achieved during T1. Subjects were continuously monitored for heart rate and SpO2 which were recorded every 2 minutes. When subjects reached 24 minutes of exercise or indicated that they wanted to stop, the treadmill was slowed to a cool-down speed of 0.7 mph for three minutes, during which time dyspnea continued to be assessed. At completion of T1, all subjects were given a pedometer and diary for monitoring home exercise and dyspnea during activities of daily living for the duration of the twelve sessions.

Results

Sample

Of the 92 patients who were initially evaluated, 33 failed to meet the inclusion criteria. Eight of the remaining subjects failed to complete the study: 4 because of hospitalization, 2 because of failure to keep appointments, 1 due to illness in his family and 1 died suddenly at home during the home walking phase of the larger study. Two subjects had missing data due to malfunctioning equipment during the stress tests and could not be included in this analysis. Therefore, 49 subjects were available for analysis at S1 and S2 .

At baseline there were no significant differences between the ME and CE group in terms of subject characteristics (Table 1). Although patients on continuous oxygen therapy were excluded, 2 patients were given oxygen per nasal cannula during treadmill exercise sessions due to desaturation to <85% during the initial ST1. There were no differences in pulmonary function after the exercise program intervention for the total sample or between treatment groups.

Total Sample

Dyspnea and Exercise Variables

Dyspnea intensity decreased significantly ($p < .01$) from a mean of 147 ± 51mm to 121 ±63mm from end exercise at baseline to end exercise after the treatment for the total

sample (N=49) (Table 3). Walking distance during the six minute walk also improved significantly ($p<.001$) (Table 3). No significant change in VE, HR, VO2, VO2/HR, or RR occurred from end exercise at baseline to end exercise after the treatment for the exercise capacity variables. To compare dyspnea and exercise capacity variables at similar workloads, an analysis was done between the baseline end exercise stage and findings at isostage after the treatment (table 3). The mean change in dyspnea for the total group (N = 49) at isostage was 59.1 ± 56 mm, with dyspnea decreasing from 147 ± 51 mm to 88± 60mm ($p<.001$). At isostage, with the exception of O2 pulse, all the exercise variables changed significantly ($p<.001$).

Relationship Between Dyspnea and Exercise Variables

Change scores were calculated for all variables by subtracting the results for each variable obtained during the isostage at stress test 2 from the results at end exercise during stress test 1. The relationship between the change in dyspnea intensity (DI) and change in exercise capacity variables was analyzed. (table 4) [see appendix 2, Operational Definitions]. For the total sample, the relationship between changes in all exercise capacity variables and changes in dyspnea was minimal and nonsignificant (Table 4).

Analysis By Severity Groups

The total sample was divided into two groups according to severity (Table 2). The standard measure of severity in pulmonary function is the FEV1 % predicted (ATS, 1991). The two groups were divided into the moderate to severe group (n=32, FEV1 % predicted <60 and ≥34%) and the very severe group (n=17, FEV1 % predicted ≤ 34%) according to American Thoracic Society criteria. There was a significant relationship between the change in some of the exercise variables and the change in dyspnea in the very severe group. The exercise capacity variables which correlated with dyspnea in this group were: HR ($r = -0.5647$, $p < 0.05$), VO2 ($r = 0.4886$, $p < 0.05$), and VO2/HR ($r = 0.6082$, $p < 0.05$).

Analysis By Treatment Group and Gender

Further analysis included an examination of the treatment group (ME and CE) and gender to determine if there was a relationship between exercise variables and dyspnea in any of the subgroups. When examined separately there was a minimal and nonsignificant relationship between exercise variables and dyspnea for males, females and for ME and CE groups.

When the sample was sorted by severity the mean change in dyspnea at end exercise stress test 2 was significant for the moderately severe group ($p < .01$), but not in the very severe group. The change in dyspnea at isostage decreased significantly more in this group ($p < .001$), with a percent change in dyspnea measuring 45% (Table 5). As in the total sample, the exercise capacity variables did not change significantly from end exercise at baseline to end exercise during stress test 2. When comparing the mean change from end exercise at baseline to isostage at ST2 in the moderate to severe group, the findings are consistent with the total sample in which DI and all the exercise capacity variables changed significantly ($p < .001$), except for O2 pulse. However, mean change for the very severe group at isostage were less significant (Table 5), and insignificant for VE and O2 pulse.

Paired T-tests were done to see if there were any differences between the two severity groups in their exercise capacity variables or perceived dyspnea at baseline end exercise. The only significant differerence was in their peak achieved HR ($p < .05$). The moderate to severe group ($n = 32$) had a peak HR of 136 ± 20, whereas the very severe group ($n = 17$) had a peak HR of 125 ± 16 (Table 5). The only significant difference between severity groups in the change scores from end exercise at baseline to isostage was for VE ($p < 0.05$).

Because the investigators questioned the highly significant correlations of the exercise capacity variables VO2, HR, and O2 pulse and the nonsignificant correlations of VE and RR with dyspnea in the very severe group (Table 4), a nonparametric correlational test (Spearman - Brown) was used to analyze the data (Table 4). Results using the

nonparametric test for the two severity groups were similar to the results obtained with Pearson R Correlation test with the exception of HR, which was still negatively correlated, but no longer statistically significant. Further analysis of the correlational findings were examined by investigating the individual changes in the exercise variables and dyspnea (Table 7).

Analysis of Individual Patterns

Four subjects from the very severely obstructed group were examined on an individual basis to describe patterns in the relationship between dyspnea and exercise capacity variables. Two women and two men, as well as two from the nurse-coached (CE) and monitored (ME) groups were chosen as exemplar cases.

Patient # 25 was a retired, divorced 60 year old male with a thin build who had a 10 year history of shortness of breath (SOB) since his diagnosis with emphysema. He had no history of other medical problems and was on no cardiac medications. He denied a history of alcohol abuse and quit smoking 15 years prior to the study. He had very severe obstructive disease, based on standardized levels of severity, (ATS, 1991). His baseline spirometry revealed an FEV1 of 0.8L, 29% predicted, with an FVC of 2.4, 60 % predicted. Pulmonary function after the exercise treatment was slightly improved with an FEV1 of 1.1L, 36% predicted and FVC of 2.55, 59%. His observed VE/MVV was 124% at baseline and 117% after the treatment which indicates that he was working above his maximum minute ventilation. It is recognized that working at ventilatory capacities near or above 100% of the MVV suggests motivated effort (Belman, 1986). After the treatment dyspnea intensity increased by 54 mm; VE, VO2, and RR decreased; and O2 pulse increased slightly at Isostage. The maximum stage reached during Stress test 2 did not change after the exercise intervention. The reason he gave for stopping the incremental stress tests was dyspnea at baseline and fatigue after the treatment.

Patient #44 was an employed, divorced 44 year old female with a thin build who had a 3 month history of SOB since her recent diagnosis of alpha1 antitrypsin also at three

months prior to the study. She quit smoking one year prior to the study. She had no other medical history and consequently was taking no cardiac medications. Her pulmonary function did not change after the exercise treatment with pre intervention values for FEV1 of .84L and 30% predicted and post FEV1 of .82L and 29% predicted. FVC changed from 66% pre to 69% post treatment. Pre treatment VE/MVV was 66% and post treatment was 57%, which may have indicated that she was either cardiac limited more than ventilatory limited, or that she was not giving the test her best effort. After the treatment, dyspnea decreased by 40 mm, with relatively no change in exercise capacity variables at isostage. She was able to complete one less stage during the stress test after the twelve sessions, therefore decreasing her exercise capacity, eventhough her dyspnea decreased. The reason for stopping the incremental stress test at baseline was dyspnea, and the reason after the treatment was "claustrophobia".

Patient # 54 was a retired, divorced, 68 year old male who described himself as "very active" and involved with "religion and spirituality". He had a history of emphysema and quit smoking four years ago. He had no other medical history and was therefore not taking any cardiac medications. His pulmonary function also did not change after the treatment with pre FEV1 of .62L, 22% predicted and post FEV1 of .74L, 29% predicted. FVC values prior to the treatment were 2.3L and 56% and after the treatment were 2.3L and 57%. Dyspnea decreased by 98mm at Isostage, while VE, VO2, HR, and RR decreased and O2 pulse increased. He was able to complete two more stages than baseline after the exercise treatment. Therefore, he decreased his dyspnea and increased his exercise capacity.

Patient #58 was a single, never married female, retired editor, age 70. She was diagnosed in 1973 with emphysema and chronic bronchitis and quit smoking at that time. She had no other diagnosed medical conditions and was not on cardiac medications. Although she was diagnosed in '73 with pulmonary disease, she denied SOB until three months prior to the study. Her pulmoanry function by spirometry slightly improved over

the course of the study with her FEV1 of .64L, 28% increasing to .81L, 36% predicted after the exercise treatment. Her FVC did not change with baseline values of 2.14L, 68% and post values of 2.24L, 71%. Dyspnea dramatically decreased by 189mm, with a significant increase in heart rate of 20 bpm at Isostage. VE, VO2, O2 pulse, and RR decreased at Isostage. She was able to complete two more stages after the exercise intervention. Therefore her dyspnea and exercise capacity both decreased. Reasons for stopping the stress tests at baseline were dyspnea/fatigue and after the treatment were "anxious at breathing".

Although these are only four of the 17 very severe subjects, there individual patterns help in the interpretation of the final results.

Discussion

The major findings of this study were that after 12 exercise sessions this sample of COPD patients at the same level of exercise dramatically decreased their perception of dyspnea and improved their exercise capacity. These results are congruent with the findings of several other investigators (Punzal et al., 1991; Strijbos et al., 1990). The improvement in exercise capacity was most probably due to increased efficiency, comfort in walking, or motivation since exercise capacity variables for the total sample did not change significantly at end exercise and the exercise intensity of the treatments was regulated by the patients. The improvement in dyspnea if not due to a training effect most probably was due to other factors, such as, repeated exposure to dyspnea during the treatments, a decrrease in anxiety related to dyspnea, changes in mood, or comfort with the environment.

More germane to the question posed in this study was the finding that there was little or no significant relationship between these changes in dyspnea and exercise capacity variables except within the very severe group. Even in that group changes in heart rate, oxygen consumption and O2 pulse were related to changes in dyspnea. This finding of a lack of a relationship between the decrease in dyspnea and the improvement in exercise

capacity when measured at isostage is interesting and corroborates the findings of others. Dyspnea has been found to correlate only moderately at best with static lung volumes and flows, such as FEV1 (Mahler, 1990). When dyspnea has been measured by interview (Cockcroft, 1981) or with scales that measure dyspnea with activity after pulmonary rehabilitation programs (Niederman et al., 1991) previous investigators have not found a relationship between reported dyspnea with activity and improvement in exercise capacity variables. Those investigators that have measured dyspnea during the exercise testing similar to this study have not reported whether they examined the relationship between the change in dyspnea and the improvement in exercise variables (Ries, et al.1994; Casaburi et. al.,1994). They only reported that both dyspnea and exercise variables improved significantly after pulmnonary rehabilitation or exercise training.

What may be the reasons for the lack of relationship between exercise capacity and dyspnea when they both improve at isostage? The lack of association may have been due to statistical problems. For example, the variation in exercise variables was small and may not have been great enough to demonstrate a significant relationship with the larger change in dyspnea. The statistical analysis was completed on only one score- a change score. In addition, the large individual variation in rating of dyspnea and changes in exercise capacity variables may have affected the correlation. Other reasons why there may be little or no relationship between decreases in dyspnea and improvement in exercise variables at the same exercise level may be the following. It is known that dyspnea is a multifactorial sensation and that the perception of shortness of breath can vary depending on many biological, environmental, and psychological factors. These could include previous repeated exposure to the sensation during the exercise sessions, comfort with walking on the treadmill, changes in breathing patterns with less hyperinflation, change in mood, and familiar environment. Many of these, or numerous other factors could change the perception of dyspnea without changing exercise capacity variables.

It is interesting that both dyspnea and exercise variables at isostage did not

improve as much in the very severe group as those in the moderate to severe group. This is congruent with what would be theoretically expected and with the findings of Niederman and colleagues (1991) who found that patients with the best lung function tended to have the greatest improvements in endurance performance. The finding that dyspnea was related to heart rate and oxygen consumption in only the very severe group is a first report of this relationship. The reason for this relationship is unknown, especially the negative relationship between dyspnea and heart rate. When analyzed individually 12 patients decreased their dyspnea and their heart rate as expected, while only one patient increased their heart rate and decreased their dyspnea. This negative relationship is primarily due to that one patient. When analyzed by a non-parametric test this relationship was no longer significant. One might have expected that if there was a relationship between any of the variables and dyspnea it would have been between minute ventilation, respiratory rate, and dyspnea. The lack of a relationship between these variables in this very severe group may have been due to the lack of change in minute ventilation, which is due to their ventilatory limitation.

The finding that there is little relationship between changes in dyspnea and exercise capacity is further evidence for the theoretical proposal that a decrease in dyspnea in patients with chronic obstructive disease may, in fact, be due to a process of desensitization. For this sample self-paced exercise and mere exposure to the symptom was a large enough stimulus to decrease dyspnea with exercise in the laboratory.

Conclusion

Because there is no definitive cure for COPD, treatment is palliative, with the goal of potentiating function and preventing decline into a debilitative state. One of the accepted methods of treatment which fosters this goal is exercise therapy. Much of the research in this area has been on COPD patients involved in pulmonary rehabilitation programs with moderate to severe obstructive disease. This study adds to the body of literature as it illustrates that even those with very severe obstruction can have profound

decreases in their dyspnea for the same level of activity when they are exercised on the treadmill. Clearly, more research is needed in this area of exercise therapy, dyspnea management and the role of desensitization which can make an impact in patient well being. This study, as well as other non pulmonary rehabilitation exercise studies, have proven that COPD patients can benefit from monitored exercise programs which emphasize symptom limitation rather than high intensities. Outcomes of improved endurance and significantly less dyspnea for similar workloads support the intervention of exercise therapy in these patients. Whether the mechanism is aerobic conditioning, increased efficiency of locomotion, desensitization to the sensation of dyspnea, or a combination of the above, the important element is if the patient is motivated to include the therapy into their lifestyle for long term benefit. Future research needs to focus on long term effects of exercise programs after either high intensity or moderate intensity in well designed, controlled studies. Research involving strategies to improve exercise compliance would also be beneficial. With the focus of health care turning toward preventative therapies, promotion of individualized exercise regimes for COPD patients based on symptom limitation and duration rather than intensity may lead to better compliance. Simple convenient exercise plans could be developed before discharge from the hospital after an exacerbation, or set up during clinic visits either individually, or in groups. Further research in this area might include strategies for improving COPD patient compliance with exercise regimes, family influence on motivation, and COPD patients belief and knowledge related to ongoing exercise therapy.

Appendix

Table of Operational Definitions

Variables	**Operational definitions**
DI (mm)	Dyspnea intensity measured on the 200mm visual analogue scale
HR (BPM)	Heart rate measured in beats per minute
VE (L/min)	Minute ventilation measured in liters per minute
VO2 (L/min)	Oxygen consumption measured in liters per minute
O2 pulse (cc/beat)	Oxygen pulse: Oxygen uptake per heart beat (VO2/HR) Oxygen pulse = stroke volume x (A-VO2) Normal: > 8ml/beat for women and > 12 ml/beat for men.
RR (bpm)	Respiratory rate measured as breaths per minute.
Baseline end exercise (ST1)	Peak exercise achieved during stress test one before the exercise intervention. This value was measured at a given stage reached at the subjects symptom-limited peak exercise. The stage workload was a given speed and grade for that stage.
Isostage	The same stage reached at baseline end exercise stress test 1 measured at stress test 2.
ST1	Baseline stress test before the exercise intervention
ST2	Stress test 2 after the exercise intervention
T1	The first treadmill test of the twelve treadmill exercise sessions (T1-T12).

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TABLE 1

SAMPLE CHARACTERISTICS

VARIABLES	Total Sample (N=49)	Moderately Severe (n=32)	Very Severe (n=17)
Gender	24 F and 25 M	20 F and 12 M	4 F and 13 M
Age (Yrs.)	66 ± 8	67 ± 8	66 ± 9
Weight (lbs.)	151 ± 38	148.7 ±32	162 ± 47
Education (Yrs.)	15 ± 3	15 ± 2	15 ± 2
Number of Yrs. Smoked (PPY)	38 ± 14	39 ± 14	36 ± 13
Years Quit Smoking (Yrs.)	10 ± 9	11 ± 10	9 ± 9
FEV1 (L)	.92 ± .24	1.0 ± 0.2	0.7 ± 0.14
FEV1% Predicted	38 ± 10	43 ± 7.7	27 ± 4.4
FVC (L)	2.3 ± 0.7	2.3 ±0.7	2.2 ± 0.6
FVC% Predicted	65 ± 15	69 ± 15	60 ± 15
FEV1/FVC%	42 ± 12	46 ± 10	35 ± 10
MVV (L/min.)	39 ± 13	42 ± 14	34 ± 10
MVV% Predicted	39 ± 11	43 ± 10	31 ± 9
VE/MVV Actual%	90 ± 20	88 ± 18	94 ± 25
ABG's			
pH	7.43 ± .03	7.44 ± .03	7.44 ± .03
PaO2 (mm Hg)	73 ± 7.9	74 ± 6	75 ± 10
PaCO2 (mmHg)	40.3 ± 5.4	40.1 ± 5	39 ± 5
Baseline Treadmill Time (min.)	8.2 ± 2.8	9.1 ± 3.0	7.4 ± 2.3

TABLE 2

CRITERIA FOR ASSESSING THE SEVERITY OF OBSTRUCTION (ATS, 1991)*f*

	FEV Percent Predicted
May be a Physiological Variant	≥100
Mild	<100 and ≥70
Moderate	<70 and ≥60
Moderately Severe *	<60 and ≥50
Severe *	<50 and ≥34
Very Severe **	<34

*Group 1 (N = 32), combination of moderately Severe and Severe subjects

**Group 2 (N = 17), very Severe subjects

f American Thoracic Society. (1991). Lung function testing: Selection of reference values and interpretive strategies. American Review of Respiratory Disease, 144, 1202-1218.

TABLE 3

ANALYSIS OF VARIANCE FOR MEAN VALUES OF EXERCISE CAPACITY AND DYSPNEA (N=49)

	BASELINE END EXERCISE	POST END EXERCISE	P	ISOSTAGE	P	FDF	% CHANGE AT ISOSTAGE
VARIABLES							
Dyspnea Intensity (200mm VAS)	147 ± 51	121 ± 63	<.01	88 ± 60	<.001	54.16	40
VE (L/min)	34 ± 10	34 ± 11	NS	27 ± 7.0	<.001	92.17	20
HR (beats/min)	130 ± 20	134 ± 18	NS	117 ± 17	<.001	38.15	10
VO2 (L/min)	.93 ± .38	.98 ± .38	NS	.80 ± .31	<.001	21.28	14
O2 Pulse (ml/beat)	7.1 ± 2.7	7.5 ± 2.4	NS	6.7 ± 2.3	NS	1.86	05
RR (breaths/min)	35 ± 6.2	34 ± 5.7	NS	29 ± 5.0	<.001	62.82	17
Stage Reached	4.59 ± 2.13	6.14 ± 2.20	<.001				

TABLE 4

RELATIONSHIP OF CHANGE SCORES IN DYSPNEA AND EXERCISE VARIABLES BETWEEN END EXERCISE (S1) AND ISOSTAGE (S2)

	Total Sample (N=49)		Moderate to Severe Group* (N=32)				Very Severe Group** (N=17)			
			Pearson's r		Spearman/Brown		Pearson's r		Spearman/Brown	
	r	P	r	P	R	P	r	P	R	P
Exercise Variables										
VE (L/min)	.2202	NS	.1469	NS	.1207	NS	.2969	NS	.2701	NS
VO_2 (L/min)	.2018	NS	.0755	NS	.0616	NS	.4886	<.05	.5490	<.05
HR (beats/min)	.0352	NS	.1640	NS	.1951	NS	-.5647	<.05	-.3632	NS
O_2 Pulse (cc/beat)	.2066	NS	.0768	NS	.0351	NS	.6082	<.05	.5882	<.05
RR (breaths/min)	.0841	NS	.1249	NS	.2340	NS	.1336	NS	-.1652	NS

* FEV1 % <60 ≥ 34%; ** FEV1 % < 34%

CHANGE SCORE FOR TOTAL SAMPLE (N = 49) [BASELINE END EXERCISE (ST1) - ISOSTAGE (ST2)]

	Change Scores	Range
DI	59.1 ± 56 SD	-61 to ± 189
VE	7.33 ± 5.3	-4.5 to +20.5
VO2	.13 ± .20	-400 to + 600
HR	14 ± 15	-27 to +50
RR	6 ± 5	-24 to + 41
O2 Pulse	.36 ± 1.7	-3.8 to +4.7

TABLE 5

MODERATE TO SEVERE GROUP (FEV_1 % <60 TO ≥34%) (n=32)

Variables	BASELINE END EXERCISE	POST END EXERCISE	P	ISOSTAGE	P	% CHANGE AT ISOSTAGE
DI (mm)	146 ± 54	117 ± 8	*.01	80 ± 56	<.001	45
VE (L/min)	36 ± 11	36 ± 10	NS	27 ± 8	<.001	22
VO_2 (L/min)	.98 ± .41	1.0 ± .41	NS	.82 ± .34	<.001	16
HR (beats/min)	136 ± 20	137 ± 19	NS	119 ± 18	<.001	13
O_2 Pulse (cc/beat)	7 ± 3	7 ± 2	NS	7 ± 3	NS	-
RR (breaths/min)	37 ± 5	35 ± 5	NS	30 ± 5	<.001	19
Treadmill Time(min)	9.1 ± 3	12.0 ± 3.6				
6 Min walk (Ft)	1401 ± 298	1538 ± 291				

VERY SEVERE GROUP (FEV_1 % <34%) (n=17)

	BASELINE END EXERCISE	POST END EXERCISE	P	ISOSTAGE	P	% CHANGE AT ISOSTAGE
DI (mm)	150 ± 44	129 ± 57	NS	103 ± 66	<.01	31
VE (L/min)	31 ± 8	33 ± 13	NS	26 ± 7	NS	16
VO_2 (L/min)	.85 ± .32	.89 ± .32	NS	.75 ± .26	<.05	12
HR (beats/min)	125 ± 16	127 ± 16	NS	115 ± 18	<.01	08
O_2 Pulse (cc/beat)	7 ± 2	8 ± 2	NS	7 ± 2	NS	-
RR (breaths/min)	32 ± 7	33± 8	NS	28 ± 5	<.001	13
Treadmill Time (min)	7.4 ± 2.3	9.0 ± 2				
6 Min Walk (ft)	13.44 ± 179	1480 ± 242				

TABLE 6

EXERCISE LIMITATION INTERPRETATION BY PERCENT PREDICTED VALUES

Groups	n	MAX HR % Pred.	MAX VO2 % Pred.	VE/MVV Act %
Moderate to Severe	32	88%	72%	88%
Very Severe	17	80%	48%	95%
Total	49	83%	64%	90%

INDIVIDUAL CHANGE SCORES FROM BASELINE TO ISOSTAGE
(VERY SEVERE GROUP; FEV1 % PREDICTED <34)

Code	Gender	TG (ME or CE)	Δ DI	Δ VE	Δ VO2	Δ HR	Δ O2 pulse	Δ RR	ST1 Peak Stage	ST2 Peak Stage	Δ Stage Reached
1	F	ME	29	2	45	14	.	.	5	4	-1
6	M	CE	54	2.5	230	11	2.0	4	1	3	+2
8	M	ME	56	9	200	4	1.5	1	3	4	+1
17	F	ME	60	0.0	29	0	0.2	5	1	2	+1
19	M	ME	73	7.0	272	9	1.4	5	5	6	+1
21	M	ME	53	6.0	219	11	1.0	5	5	6	+1
22	M	ME	71	2.0	166	15	0.4	-1	5	4	-1
25*	M	CE	-54	2.5	12	10	-0.4	8	4	4	NO Δ
27	M	CE	7	-1.0	-227	7	-2.2	13	3	4	+1
34	M	ME	-21	7.0	140	19	0.4	1	3	4	+1
39	M	ME	3	4.8	60	14	-0.2	10	4	6	+2
44*	F	ME	40	1.4	-30	1	-0.3	-1	6	5	-1
45	M	CE	-29	6.0	120	17	-0.2	1	5	6	+1
51	M	ME	17	11.7	-120	18	0.2	7	4	5	+1
52	M	CE	155	10.5	385	12	2.1	4	5	8	+3
54*	M	CE	98	8.4	65	16	-0.8	7	4	6	+2
58*	F	ME	189	6.5	150	-20	2.3	5	4	6	+2

*Four individuals selected for individual analysis

Direction of Change For Individual Scores

code	ΔDI	ΔVE	ΔVO2	ΔHR	ΔO2Pulse	ΔRR	ΔStages
#25	↑54	↓2.5	↓.012	↓10	0.4↑	↓8	no Δ
#44	↓40	↓1.4	↑.030	↓1	↑0.3	↑1	↓1
#54	↓98	↓8.4	↓.065	↓16	↑0.8	↓7	↑2
#58	↓189	↓6.5	↓.150	↑20	↓2.3	↓5	↑2