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Effect of Treadmill Exercise Training on VO_2 Peak in Chronic Obstructive Pulmonary Disease

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ABSTRACT

Background: *Impairment of exercise tolerance in chronic respiratory disorders and in particular, chronic obstructive pulmonary disease (COPD), has important implications on health-related quality of life, hospitalization rate and survival. Although COPD patients have shortness of breath, programmed exercise can increase the activity tolerance in these patients. Maximal oxygen uptake (VO_2 peak) is a fundamental measure of exercise physiology. It is an index of cardiovascular performance as well as a measure of aerobic capacity. VO_2 max can be measured by expensive devices or predicted by a valid formula. The objective of this study was to determine the effect of treadmill exercise training (TET) on patients with chronic obstructive pulmonary disease after a 4-week program.*

Materials and Methods: *Thirty-eight subjects with COPD were recruited in the study and were randomly divided into two groups of control (n=18) and treatment (TET, n=20). The control group did not do any treadmill exercise training (TET). The treatment group exercised on a treadmill three times a week. VO_2 max was assessed in each group before and after the training period by using a modified formula.*

Results: *After 4 weeks of training, VO_2 peak was significantly greater in the TET group.*

Conclusion: *Considering the increase in VO_2 peak, It was concluded that TET benefits exercise performance, inspiratory muscle strength, dyspnea and health-related quality of life.* ***(Tanaffos 2007; 6(4): 18-24)***

Key words: *Activity intolerance, VO_2 peak, VO_2 max, Exercise training, Rehabilitation, COPD*

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is the only cause of death among the list of chronic diseases predicted to increase worldwide up to 2020 (1). It poses a big burden on the society (2-5). Respiratory rehabilitation including the cessation of smoking, psychological support, and physical exercise improve exercise capacity and health-related quality of life (HRQL) (6) and are cost effective (7).

COPD is a major cause of morbidity, mortality and health care costs (8). Exercise intolerance is one of the most troublesome manifestations of COPD.

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Patients in initial stages of the disease may experience dyspnea during heavy exertion that is attributed to "slowing down with age." Patients with moderate and severe COPD commonly have difficulty performing normal daily tasks such as work, recreational exercise, hobbies, and self-care. Dyspnea, leg fatigue, and discomfort are the main symptoms that limit exercise (9), and patients typically limit their activities to avoid these uncomfortable symptoms. The resultant inactivity leads to a progressive deterioration that further increases the respiratory effort related to any given task. Ultimately, patients often become progressively homebound and isolated, and may develop worsening depression and anxiety. Such depression is associated with significant disturbances in physical function (10). Indeed, exercise capacity and health status also correlate inversely with morbidity (11).

The management of COPD has advanced significantly over the past few years. In particular, pulmonary rehabilitation has gradually been accepted as the gold standard of care for patients with COPD. Recent evidence-based guidelines (12) have considered exercise training a necessary and critically important part of pulmonary rehabilitation if desired results are to be achieved.

Over the recent years, researchers and clinicians have increasingly recognised the role of skeletal muscle dysfunction as an indicator of advanced stages of COPD. The physical exercise component has therefore become a mainstay of respiratory rehabilitation. Several studies have shown that physical exercise can reverse COPD induced skeletal muscle dysfunction as well as the morphological and metabolic changes of skeletal muscles. There is; however, substantial variation in exercise protocols used in clinical trials. This variation feeds an ongoing debate on the optimal exercise protocol and on how the general impact of modifying principles, training intensity, specificity, and reversibility known from healthy subjects can be applied to COPD patients. Trial results are conflicting with regard to the intensity which COPD patients should exercise, how the intensity should be determined, and whether continuous or interval exercise is appropriate (13).

VO_2 max is the maximum volume of oxygen that the body can consume during intense, whole-body exercise, while breathing at sea level. This volume is expressed as a rate, either litre per minute (L/min) or millilitre per kg bodyweight per minute (ml/kg/min). Since oxygen consumption is linearly related to energy expenditure, when we measure oxygen consumption, we are indirectly measuring an individual's maximal capacity to do work aerobically. VO_2 max calculation is used in healthy persons and athletes. In patients who may not obtain the maximum volume of oxygen the body can consume during intense exercise, VO_2 peak can be used instead of VO_2 max.

Maximal oxygen uptake (VO_2 max or VO_2 peak) is a fundamental measure of exercise physiology. It is an index of cardiovascular performance as well as a measure of aerobic capacity. VO_2 max can be calculated by expensive devices or predicted by a valid formula (14) and specific protocols, such as the Bruce protocol (15).

Since both aerobic endurance training and weight training can be beneficial and safe for patients with COPD (when done properly), most rehabilitation programs currently use both types of training. There is controversy regarding the mechanisms of improvement attributable to exercise training, and the optimal exercise guidelines for exercise training in COPD patients cannot be defined with certainty (16).

Given the limited research on optimal exercise guidelines for exercise training in COPD patients, the purpose of this study was to evaluate the effect of treadmill exercise training program (TET) after 4

weeks on VO_2 peak in chronic obstructive pulmonary disease patients.

MATERIALS AND METHODS

Thirty-eight COPD patients were enrolled in the study and were randomly divided into two groups: the control group (n=18) and the treatment group (n=20). In this randomized controlled clinical trial (semiexperimental design), the control group did not do any treadmill exercise training (TET). The treatment group exercised on a treadmill in the clinic, 2 times a week for 8 weeks. As the patient ran on the electrical treadmill (High-life Model A – 340, Taiwan) to exhaustion, vital signs were evaluated by digital monitoring devices. The treadmill was set at a minimum speed and 0° elevation. First, the patients warmed up by performing stretched exercises for 5 minutes and then commenced exercise. The speed was gradually increased according to the patient's ability and tolerance. He/she was under complete supervision, and the training program was stopped upon the patient's request or critical changes in vital signs. All patients were given complete instructions on the testing procedure before signing a written informed consent. Each subject arrived 1 hour prior to the exercise for instructions and familiarization. Prior to the familiarization process, subjects gave an informed consent and filled out a medical history questionnaire. Additionally, data including age (yrs), height (cm), weight (kg), resting heart rate (beats/min), resting blood pressure (mmHg) and smoking experience (years) were collected. Severity of COPD was estimated by using spirometric parameters. If 50%≤ FEV1<80% and FEV1/ FVC<0 the patient had moderate, and if 30%≤ FEV1<50% and FEV1/ FVC< 0.7 the patient had severe COPD.

In order to determine VO_2 peak, the patient should run on a 1 mile distance and the time duration of 1 mile running was recorded and entered in the Rockport formula. Due to the inability of COPD patients to complete the 1 mile distance, the duration of running any distance was calculated instead of a mile proportionally. Therefore, in each group VO_2 peak, was assessed via a modified Rockport formula before and after 8 weeks of training. The calculated VO_2 peak is not equal to actual VO_2 peak, therefore, we determined the change of VO_2 peak. During the exercise test, no oxygen was administered to the subjects of either group.

The formula used to calculate the VO_2 max is:

- 132.853 - (0.0769 × Weight) - (0.3877 × Age) + (6.315 × Gender) - (3.2649 × Time) - (0.1565 × Heart rate)

Where: Weight is in pounds (lbs), Gender Male = 1 and Female = 0, Time is expressed in minutes and 100ths of a minute, Heart rate is in beats/minute, Age is in years.

RESULTS

According to data analysis, there was no significant difference in the mean age between the control group (7 males and 11 females; 52.17 ± 11.6 yrs.) and the treatment group (11 males and 9 females; 52.1 ± 10.7 yrs.). Of 38 participants, 24 subjects (63.15%) had past history of COPD for ≤10 years. More than 80% of subjects had severe COPD (30%≤ FEV1<50% and FEV1/ FVC< 0.7), and there was no difference between the two groups. Fifteen subjects (83.3%) in the control group and 18 subjects (90%) in the treatment group were in severe while the remaining were in mild states of the disease. During the study period, no changes were made in the prescribed drugs. All subjects were using bronchodilators and at least 30% of them were using corticosteroids.

Table 1 summarizes the effect of treadmill exercise training on VO_2 peak in both groups. It can be seen in this table that the mean VO_2 peak before the training program had no significant difference in

the control and treatment subjects. But, the mean VO_2 peak after the training program in the treatment group (Mean 31.73 SD ± 6.46) was significantly ($\rho<0.001$) higher than the control group (mean ± SD 18.01± 7.04). Table 1 shows that a significant change in VO_2 peak was only seen in the treatment group.

Table 1. The comparison of VO_2 peak before and after treadmill exercise training between the two groups and in each group alone.

VO_2 peak (ml/kg/min) Groups	Before (*Mean ±SD*)	After (*Mean ±SD*)
control group *(n=18)*	18.35 ± 7.28	18.01 ± 7.04
Treatment group (n=20)	16.42 ± 6.74	31.73 ± 6.46*

** Significant P<0.001*

Table 2. The comparison of heart rate, running distance and time of treadmill exercise training before and after treadmill exercise training in each group.

Variables	Control group (n=18)		Treatment group (n=20)	
	Before (Mean ±SD)	After (Mean ±SD)	Before (Mean ±SD)	After (Mean ±SD)
Heart rate (beats/min)	135.39±19.87	129.11± 35	127±16.24	115.±16.479*
Running distance (meter)	330 ±146.2	318.3±134.3	343± 207	1151.5±338*
Time of running (minutes)	9.47 ± 2.94	6.19± 2.55	6.98± 4.27	16.37±4.83*

**Significant P<0.001*

DISCUSSION

Although some studies have shown improvements in VO_2 peak after exercise training in chronic heart failure patients (17-19), others have not (20-22). Vallet et al. (23) assessed whether exercise at the individual anaerobic threshold was more effective than exercise at 50% of the maximal heart rate reserve (standardized protocol). Mean exercise intensities between the two groups were not different. The authors reported a trend towards larger increases of peak oxygen uptake and anaerobic threshold with the individualized exercise protocol. There was a significantly greater training response in the group with the individualized protocol in terms of reductions in minute ventilation, lactate levels, and carbon dioxide output at given levels of pre-training oxygen uptake. Among patients with moderate to severe COPD, the factors which can distinguish patients who most likely respond to exercise training are not completely understood. One study found that patients with severe COPD and severe dyspnea (assessed by the Medical Research Council dyspnea score) at baseline did not achieve significant improvements in walking distance following training (24), but several other studies have shown that even COPD patients with severe dyspnea can achieve gains in exercise tolerance by exercise training (25-27).

In our study, significant improvements were found in VO_2 peak, time of exercise, running distance and reduction of heart rate after exercise. Skeletal muscle dysfunction in COPD results from reductions in muscle mass and strength (28,29), atrophy of type I (slow twitch, oxidative, endurance) (30,31) and type IIa (fast-twitch, glycolytic) muscle fibers (32), reduction in fiber capillarization (33) and oxidative enzyme capacity (34, 35), reduced muscle endurance (30,31), resting and exercise impaired muscle metabolism (36,37,38), lactic acidosis (39,40) and hemodynamic changes (17,19). Our finding implied that training did affect muscle metabolism and production of lactic acid and resulted in hemodynamic improvement.

In conclusion, the greater activity tolerance and lower measured physiological responses achieved by training exercises may allow fatigue adaptations in

individuals with limited lung function. The results suggest that programmed exercise may be useful as a mode of rehabilitation in patients with COPD. It seems that TET directly or indirectly yields great benefits in improving exercise performance, inspiratory muscle strength, dyspnea and health-related quality of life.

Further studies are necessary to evaluate how ventilation improvement can be achieved, and how long this can be maintained in a steady state.

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REFERENCES

1. World Health Organisation. World health report 2000. Geneva: World Health Organisation, 2000, www.who.int/whr/2000/en/statistics.htm.
2. Seemungal TA, Donaldson GC, Paul EA, Bestall JC, Jeffries DJ, Wedzicha JA. Effect of exacerbation on quality of life in patients with chronic obstructive pulmonary disease. ***Am J Respir Crit Care Med*** 1998; 157 (5 Pt 1): 1418- 22.
3. Sin DD, Stafinski T, Ng YC, Bell NR, Jacobs P. The impact of chronic obstructive pulmonary disease on work loss in the United States. ***Am J Respir Crit Care Med*** 2002; 165 (5): 704- 7.
4. Sullivan SD, Ramsey SD, Lee TA. The economic burden of COPD. ***Chest*** 2000; 117 (2 Suppl): 5S- 9S.
5. Decramer M, Gosselink R, Troosters T, Verschueren M, Evers G. Muscle weakness is related to utilization of health care resources in COPD patients. ***Eur Respir J*** 1997; 10 (2): 417- 23.
6. Lacasse Y, Brosseau L, Milne S, Martin S, Wong E, Guyatt GH, et al. Pulmonary rehabilitation for chronic obstructive pulmonary disease. ***Cochrane Database Syst Rev*** 2002; (3): CD003793. Update in: ***Cochrane Database Syst Rev*** 2006; (4): CD003793.
7. Griffiths TL, Phillips CJ, Davies S, Burr ML, Campbell IA. Cost effectiveness of an outpatient multidisciplinary pulmonary rehabilitation programme. ***Thorax*** 2001; 56 (10): 779- 84.
8. Mannino DM. COPD: epidemiology, prevalence, morbidity and mortality, and disease heterogeneity. ***Chest*** 2002; 121 (5 Suppl): 121S- 126S.
9. Killian KJ, Leblanc P, Martin DH, Summers E, Jones NL, Campbell EJ. Exercise capacity and ventilatory, circulatory, and symptom limitation in patients with chronic airflow limitation. ***Am Rev Respir Dis*** 1992; 146 (4): 935- 40.
10. Lacasse Y, Rousseau L, Maltais F. Prevalence of depressive symptoms and depression in patients with severe oxygen-dependent chronic obstructive pulmonary disease. ***J Cardiopulm Rehabil*** 2001; 21 (2): 80- 6.
11. Oga T, Nishimura K, Tsukino M, Sato S, Hajiro T. Analysis of the factors related to mortality in chronic obstructive pulmonary disease: role of exercise capacity and health status. ***Am J Respir Crit Care Med*** 2003; 167 (4): 544- 9.
12. Pulmonary rehabilitation: joint ACCP/AACVPR evidence-based guidelines. ACCP/AACVPR Pulmonary Rehabilitation Guidelines Panel. American College of Chest Physicians. American Association of Cardiovascular and Pulmonary Rehabilitation. ***Chest*** 1997; 112 (5): 1363- 96.
13. Puhan MA, Schünemann HJ, Frey M, Scharplatz M, Bachmann LM. How should COPD patients exercise during respiratory rehabilitation? Comparison of exercise modalities and intensities to treat skeletal muscle dysfunction. ***Thorax*** 2005; 60 (5): 367- 75.
14. Buchfuhrer MJ, Hansen JE, Robinson TE, Sue DY, Wasserman K, Whipp BJ. Optimizing the exercise protocol for cardiopulmonary assessment. ***J Appl Physiol*** 1983; 55 (5): 1558- 64.
15. Najafi MS, Najafi Kaliani M, Ghasemi A, Ebadi A, Tayebi A, Hafezi S. The comparison of Milad NBC Protective Clothing without mask and military work clothes on maximal

oxygen consumption (VO_2 max). ***Kosar Medical Journal*** 2006; 11(4): 367-72.

16. American College of Sports Medicine. Guidelines for Exercise Training and Prescription. 5th ed. Baltimore: Williams & Wilkins; 1995.
17. Coats AJ, Adamopoulos S, Radaelli A, McCance A, Meyer TE, Bernardi L, et al. Controlled trial of physical training in chronic heart failure. Exercise performance, hemodynamics, ventilation, and autonomic function. ***Circulation*** 1992; 85 (6): 2119- 31.
18. Barlow CW, Qayyum MS, Davey PP, Conway J, Paterson DJ, Robbins PA. Effect of physical training on exercise-induced hyperkalemia in chronic heart failure. Relation with ventilation and catecholamines. ***Circulation*** 1994; 89 (3): 1144- 52.
19. Belardinelli R, Georgiou D, Cianci G, Berman N, Ginzton L, Purcaro A. Exercise training improves left ventricular diastolic filling in patients with dilated cardiomyopathy. Clinical and prognostic implications. ***Circulation*** 1995; 91 (11): 2775- 84.
20. Belardinelli R, Georgiou D, Cianci G, Purcaro A. Randomized, controlled trial of long-term moderate exercise training in chronic heart failure: effects on functional capacity, quality of life, and clinical outcome. ***Circulation*** 1999; 99 (9): 1173- 82.
21. Wilson JR, Groves J, Rayos G. Circulatory status and response to cardiac rehabilitation in patients with heart failure. ***Circulation*** 1996; 94 (7): 1567- 72.
22. Willenheimer R, Erhardt L, Cline C, Rydberg E, Israelsson B. Exercise training in heart failure improves quality of life and exercise capacity. ***Eur Heart J*** 1998; 19 (5): 774- 81.
23. Vallet G, Ahmaïdi S, Serres I, Fabre C, Bourgouin D, Desplan J, et al. Comparison of two training programmes in chronic airway limitation patients: standardized versus individualized protocols. ***Eur Respir J*** 1997; 10 (1): 114- 22.
24. Wedzicha JA, Bestall JC, Garrod R, Garnham R, Paul EA, Jones PW. Randomized controlled trial of pulmonary rehabilitation in severe chronic obstructive pulmonary disease patients, stratified with the MRC dyspnoea scale. ***Eur Respir J*** 1998; 12 (2): 363- 9.
25. Maltais F, LeBlanc P, Simard C, Jobin J, Bérubé C, Bruneau J, et al. Skeletal muscle adaptation to endurance training in patients with chronic obstructive pulmonary disease. ***Am J Respir Crit Care Med*** 1996; 154 (2 Pt 1): 442- 7.
26. Goldstein RS, Gort EH, Stubbing D, Avendano MA, Guyatt GH. Randomised controlled trial of respiratory rehabilitation. ***Lancet*** 1994; 344 (8934): 1394- 7.
27. Votto J, Bowen J, Scalise P, Wollschlager C, ZuWallack R. Short-stay comprehensive inpatient pulmonary rehabilitation for advanced chronic obstructive pulmonary disease. ***Arch Phys Med Rehabil*** 1996; 77 (11): 1115- 8.
28. Baarends EM, Schols AM, Mostert R, Wouters EF. Peak exercise response in relation to tissue depletion in patients with chronic obstructive pulmonary disease. ***Eur Respir J*** 1997; 10 (12): 2807- 13.
29. Bernard S, LeBlanc P, Whittom F, Carrier G, Jobin J, Belleau R, et al. Peripheral muscle weakness in patients with chronic obstructive pulmonary disease. ***Am J Respir Crit Care Med*** 1998; 158 (2): 629- 34.
30. Hildebrand IL, Sylvén C, Esbjörnsson M, Hellström K, Jansson E. Does chronic hypoxaemia induce transformations of fibre types? ***Acta Physiol Scand*** 1991; 141 (3): 435- 9.
31. Jakobsson P, Jorfeldt L, Brundin A. Skeletal muscle metabolites and fibre types in patients with advanced chronic obstructive pulmonary disease (COPD), with and without chronic respiratory failure. ***Eur Respir J*** 1990; 3 (2): 192- 6.
32. Hughes RL, Katz H, Sahgal V, Campbell JA, Hartz R, Shields TW. Fiber size and energy metabolites in five separate muscles from patients with chronic obstructive lung diseases. ***Respiration*** 1983; 44 (5): 321- 8.
33. Simard C, Maltais F, LeBlanc P, Simard M, Jobin J. Mitochondrial and capillarity changes in the vastus lateralis muscle of COPD patients: electron microscopy study. ***Med Sci Sports Exerc*** 1996; 28: 95S.
34. Jakobsson P, Jorfeldt L, Henriksson J. Metabolic enzyme activity in the quadriceps femoris muscle in patients with severe chronic obstructive pulmonary disease. ***Am J Respir Crit Care Med*** 1995; 151 (2 Pt 1): 374- 7.
35. Gosker HR, van Mameren H, van Dijk PJ, Engelen MP, van der Vusse GJ, Wouters EF, et al. Skeletal muscle fibre-type

shifting and metabolic profile in patients with chronic obstructive pulmonary disease. ***Eur Respir J*** 2002; 19 (4): 617- 25.

36. Maltais F, Simard AA, Simard C, Jobin J, Desgagnés P, LeBlanc P. Oxidative capacity of the skeletal muscle and lactic acid kinetics during exercise in normal subjects and in patients with COPD. ***Am J Respir Crit Care Med*** 1996; 153 (1): 288- 93.
37. Maltais F, Jobin J, Sullivan MJ, Bernard S, Whittom F, Killian KJ, et al. Metabolic and hemodynamic responses of lower limb during exercise in patients with COPD. ***J Appl Physiol*** 1998; 84 (5): 1573- 80.
38. Engelen MP, Schols AM, Does JD, Gosker HR, Deutz NE, Wouters EF. Exercise-induced lactate increase in relation to muscle substrates in patients with chronic obstructive pulmonary disease. ***Am J Respir Crit Care Med*** 2000; 162 (5): 1697- 704.
39. Polkey MI. Muscle metabolism and exercise tolerance in COPD. ***Chest*** 2002; 121 (5 Suppl): 131S- 135S.
40. Casaburi R. Skeletal muscle dysfunction in chronic obstructive pulmonary disease. ***Med Sci Sports Exerc*** 2001; 33 (7 Suppl): S662- 70.