CLINICAL REVIEW

REHABILITATION FOR CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS - EXERCISE TRAINING COMPONENT

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Introduction

Even though Barach in 1964 advocated physical exercise for patients with chronic lung diseases (1), it was only in early 1970s that a liberal use of exercise training was included in pulmonary rehabilitation programmes (2). The relentless downhill course of chronic obstructive pulmonary disease (COPD) over many years and the concomitant worsening of dyspnoea limit the activity of patients, leading to a vicious cycle of increasing inactivity and dyspnoea. This in turn aggravates the debilitating effects of the disease. Exercise training has been advocated as an important component in pulmonary rehabilitation to improve well-being and to reduce subsequent hospital admissions in patients with chronic obstructive pulmonary disease.

Exercise pathophysiology in COPD

Expiratory airflow obstruction is the main physiological abnormally in COPD. In moderate to severe COPD, resting expiratory airflows approach are equal to maximal airflow. In patients with severe disease, flow limitation is present even at rest. As a result, expiratory air flow limitation occurs in patients with COPD during exercise, but not in normal subjects. The prolongation of expiration together with a higher than normal minute ventilation (VE) during exercise leads to dynamic hyperinflation with an increase in end expiratory lung volume in COPD (3,4,5). In moderate to severe COPD, maximal exercise ventilation (VEmax) reaches a high percentage of the maximal Voluntary ventilation(MVV) measured at rest. Therefore VEmax/MVV ratio may even exceed 100% in severe COPD during exercise, whereas in normal subjects the VE max/MVV ratio is less than 75% (6).

Hypoxaemia, hypercapnia, acidaemia and malnutrition which are frequently observed in patients with COPD impair respiratory muscle contractility. Diaphragm is the principal muscle of respiration and hyperinflation and flattening of the diaphragm lead to diminished inspiratory force in COPD. A significant decrease in the maximal inspiratory pressure (PImax) had been demonstrated in COPD. Because of altered respiratory mechanics, patients with COPD have to generate greater inspiratory pressure and this may lead to inspiratory muscle fatigue during exercise (7,8,9).

Maximal oxygen consumption (VO_2 max) and Oxygen-pulse (oxygen consumption per heartbeat) at maximum exercise are decreased in patients with COPD. The reduced VO_2 max during exercise is due to impaired ventilatory or diffusing capacity or both. Hypoxaemia, a common feature of emphysematous type COPD at rest, frequently shows further reductions during exercise. A decrease in resting diffusing capacity below 55 percent of predicted had been shown to be a good predicator of arterial desaturation in patients with COPD during exercise, However, some patients may show an improvement in arterial oxygen tension (PaO_2) with exercise which might be due to an improvement in pulmonary ventilation perfusion matching (10,11). The ratio of physiologic dead space to tidal volume is increased in COPD and remains high during exercise, leading to an increase in $PaCO_2$ in patients with moderate to severe COPD.

Elevated pulmonary vascular pressure and resistance which are frequent findings in COPD result from remodelling of the muscular arteries and arterioles, emphysematous destruction of the vascular bed, alveolar hypoxia, increased alveolar pressure, increased haematocrit and acidosis. In normal subjects exercise cakes a minor increase in pulmonary artery pressure. However, in subjects with COPD there is an inappropriate elevation of pulmonary artery pressure with exercise. Normally both right and left ventricular ejection fractions increase by five percent during exercise. But in patients with COPD, an abnormal right ventricular ejection fraction response (< 5 percent increase) and a normal left ventricular ejection fraction response (> 5 percent increase) occur during exercise (3,12,13).

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COPD patients may develop metabolic acidosis at end exercise. The point at which blood lactate rises during incremental exercise has been termed as lactic acid threshold. The lactic acid threshold precedes the increase in minute ventilation and the increased carbon dioxide (VCO_2) output. While oxygen uptake remains linear at the onset of lactic acid production, Co_2 output increases. As a result, if CO_2 output is plotted against O_2 uptake, VCO_2 accelerates compared to VO_2 and this inflection point is termed as the 'anaerobic threshold' (14, 15). The anaerobic threshold has been detected noninvasively by gas exchange measurements in patients with COPD. A significant number of patients with COPD stop exercising because of peripheral muscle fatigue (16).

Exercise training

It had only been recently confirmed in a randomized study that exercise reconditioning is an essential component of the rehabilitation process in COPD. In this study, the patients in the treated group were provided education, physical and respiratory therapy, psychosocial support and supervised exercise training while patients in the control group received twice weekly class room instruction in respiratory therapy, lung disease, pharmacology and diet but without exercise. At eight weeks, exercise endurance as measured by treadmill walking showed a mean increase in treadmill time from 12.5 minutes to 23 minutes in treated group compared with an insignificant change from 12 to 13 minutes in the control group (17). Various other studies had also confirmed the usefulness of exercise training in COPD (18, 19). It had been suggested that aerobic training in COPD would reduce CO_2 output and ventilatory stimulus. The lactic acid produced during exercise is buffered mainly by bicarbonate with the generation of carbonic acid which dissociates into CO_2 and H_2O. Thus Co, produced by buffering of lactic acid and produced by muscle metabolism during exercise is excreted by the lungs. Exercise training delays the rise in blood lactate levels and reduces CO_2 load; thus decreasing ventilatory requirements during exercise. The effect of aerobic training and reduction in ventilation (VE) during exercise has been well documented in normal subjects (20,21). Based on these principles, when high and low intensity training were performed in patients with COPD, a reduction in peak carbon dioxide production (VCO_2) and the maximal ventilatory equivalent for oxygen (VE/VO_2) were noticed in high intensity group. The high intensity trained group showed significant reductions in blood lactate, ventilation (VE), Carbon dioxide output (VCO_2), Oxygen consumption (VO_2) and the VE/VO_2 ratio. Heart rate at comparable work rates was reduced. A significant increase in endurance exercise was also seen in the high intensity trained group (21). However, studies had demonstrated that there is no intrinsic benefits in insisting that training be performed at almost maximal ventilatory capacity in patients with COPD (22). It had also been demonstrated that even severely obstructed patients can be exercised safely and show impressive gains in submaximal exercise endurance (23).

Physical training consists of exercise training, respiratory resistive loading and breathing training (11). Exercise training programme in general include stair climbing, walking, exercise on a treadmill or bicycle ergometer (11,24). The simplest form of exercise is walking which can be performed for a definite period of time (three, six or 12 minutes depending on the tolerance of the patient). Both speed of walking and duration of each exercise period can be increased gradually. A health professional should monitor the exercise sessions so that the patient can continue the programme without fear and anxieties in the house. Most activities of daily living such as washing, combing hair and brushing teeth cause dyspnoea in patients with severe COPD. An upper extremity exercise programme such as lifting a cane or stick from wrist level to above the head may improve the ability of these patients to perform these activities without dyspnoea. Maximum exercise ventilation and oxygen consumption improve in most patients with exercise training.

Reduced ventilatory capacity is the major limiting factor in exercise in patients with COPD and therefore, exercise to strengthen respiratory muscles may be beneficial. In conformity with this, it had been demonstrated that training respiratory muscles with an inspiratory resistive device had resulted in an increase in maximum oxygen uptake and minute ventilation (25).

Breathing training that included pursed-lip breathing, expiratory abdominal augmentation, synchronization of movement of abdomen and thorax, relaxation techniques for the accessory respiratory muscles, psychological assurance and education of patient about the disease had also been shown to improve exercise performance in patients with COPD (26). In pursed-lip breathing, patient is instructed to inspire slowly for two seconds, then breathe out slowly for approximately 10 seconds against a mild resistance created by gently pursing the lip. Pursed lip breathing had been shown to increase tidal volume, decrease respiratory rate and minute ventilation and decrease $PaCO_2$.

Mechanisms of improvement

Major components of the aerobic training responses in normal subjects are increased capillary and mitochondrial density together with increased concentrations of oxidative enzymes in trained muscles (27). However, muscle biopsies from trained limbs of COPD patients have not demonstrated improvement in oxidative enzymes (28). This may be due to the fact that COPD patients are unable to exercise at the threshold intensity necessary to elicit a true aerobic response. A third of patients with COPD had implicated muscle fatigue as the limiting factor during exercise. Therefore, strength training should form an important component of the training programme (29). Increased motivation can be an important factor for the improvement especially in submaximal steady state exercise tests,

Various studies using scales and questionnaires including the Borg scale for perceived exertion and the chronic respiratory questionnaire had demonstrated improvements in well being and reduction in breathlessness following exercise training (30,31). There are evidences that desensitization to dyspnoea may play a part after exercise. When patients with dyspnoea are trained in a medically controlled environment, while simultaneously receiving support and encouragement, they learn to overcome the anxiety and apprehension associated with dyspnoea (9). It had been demonstrated that skill in treadmill walking improved with repeated attempts. Skillful performance of the task decreases both the oxygen cost and the ventilatory requirements of work (32). It had also been observed that there was no appreciable change In pulmonary function and gas exchange following exercise in patients with COPD.

Studies, especially from our country, are required to evaluate the role of yoga exercise in pulmonary rehabilitation of patients with COPD.

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