

**COMPARATIVE ANALYSIS OF UPPER LIMB AND LOWER LIMB  
EXERCISE TRAINING ALONG WITH CONVENTIONAL  
PHYSIOTHERAPY IN IMPROVING FEV1 AND VITAL  
CAPACITY IN COPD SUBJECTS**

*A dissertation submitted in partial fulfillment of the requirement for the degree of*

**MASTER OF PHYSIOTHERAPY  
ELECTIVE – ADVANCED PT IN CARDIO RESPIRATORY**

**To**

**The Tamil Nadu Dr. M.G.R. Medical University**

**Chennai-600012**

**April 2012**



**Reg No: 27101915**

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**INTERNAL EXAMINER:**

**EXTERNAL EXAMINER:**

**SUBMITTED IN THE PARTIAL FULFILLMENT OF THE REQUIREMENT**

**FOR DEGREE OF “MASTER OF PHYSIOTHERAPY”**

**TO**

**THE TAMIL NADU**

**DR. M.G.R. MEDICAL UNIVERSITY,**

**CHENNAI.**

**APRIL 2012**

## **DECLARATION**

I hereby declare and present my project work entitled **“COMPARATIVE ANALYSIS OF UPPER LIMB AND LOWER LIMB EXERCISE TRAINING ALONG WITH CONVENTIONAL PHYSIOTHERAPY IN IMPROVING FEV1 AND VITAL CAPACITY IN COPD SUBJECTS”** The outcome of the original research work undertaken and carried out by me, under the guidance of Professor Mrs.S.Seema ,MPT ,RVS college of physiotherapy, Sulur, Coimbatore.

I also declare that the material of this project work has not formed in any way the basis for the award of any other degree previously from the Tamil Nadu Dr. M.G.R Medical University.

Date :

SIGNATURE

Place :

## **ACKNOWLEDGEMENT**

I magnify the LORD MY GOD, I will give thanks with my whole heart will tell of your wonderful deeds now and forever.

I humbly acknowledge all the love and care showered by the parents Mr. K K Kuriakose & Mrs. Sophy Kuriakose throughout my life in making me what I am.

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## I.INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a major cause of morbidity, mortality, and health care use. Exercise intolerance is one of the most troubling manifestations of COPD. Chronic Obstructive Pulmonary Disease (COPD) is a chronic slowly progressive disorder characterized by airflow obstruction (reduced FEV1 and FEV1/VC ratio) that does not change markedly over several months. In India a median prevalence of 5 per cent in men and 2.7 per cent in women was calculated which accounted for a total burden of 8.15 million male and 4.21 million female patients in a population of 944.5 million in 1996.

The American Thoracic Society and European Respiratory Society have defined pulmonary rehabilitation as: “an evidence-based, multidisciplinary, and comprehensive intervention for patients with chronic respiratory diseases who are symptomatic and often have decreased daily life activities. Integrated into the individualized treatment of the patient, pulmonary rehabilitation is designed to reduce symptoms, optimize functional status, increase participation, and reduce health care costs through stabilizing or reversing systemic manifestations of the disease”

Chronic obstructive pulmonary disease (COPD), also known as chronic obstructive lung disease (COLD), chronic obstructive airway disease (COAD), chronic airflow limitation (CAL) and chronic obstructive respiratory disease (CORD), is the co-occurrence of chronic bronchitis and emphysema, a pair of commonly co-existing diseases of the lungs in which the airways become narrowed. This leads to a limitation of the flow of air to and from the lungs, causing shortness of breath (dyspnea). In clinical practice, COPD is defined by its

characteristically low airflow on lung function tests. In contrast to asthma; this limitation is poorly reversible and usually gets progressively worse over time.

COPD is caused by noxious particles or gas, most commonly from tobacco smoking, which triggers an abnormal inflammatory response in the lung. The diagnosis of COPD requires lung function tests. Important management strategies are smoking cessation, vaccinations, rehabilitation, and drug therapy. Some patients go on to require long-term oxygen therapy or lung transplantation. Worldwide, COPD ranked as the sixth leading cause of death in 1990. It is projected to be the fourth leading cause of death worldwide by 2030 due to an increase in smoking rates and demographic changes in many countries

Chronic obstructive pulmonary disease, or COPD, is a long-lasting obstruction of the airways that occurs with chronic bronchitis, emphysema, or both. This obstruction of airflow is progressive in that it happens over time. Chronic bronchitis is defined as a chronic cough not caused by another condition that produces sputum for 3 or more months during each of the 2 consecutive years. In chronic bronchitis, the mucous glands in the lungs become larger. The airways become inflamed, and the bronchial walls thicken. These changes and the loss of supporting alveolar attachments limit airflow by allowing the airway walls to deform and narrow the airway lumen. Emphysema is an abnormal, permanent enlargement of the air spaces located at the end of the breathing passages of the lungs. Emphysema also destroys the walls of these air spaces. There are 3 types of emphysema: centriacinar emphysema, panacinar emphysema, and distal acinar emphysema or

paraseptal emphysema. Cigarette smoking or exposure to tobacco smoke is the primary cause of COPD.

While COPD occurs in 15% of cigarette smokers, tobacco use accounts for as much as 90% of the risk for the development of this disease. Secondhand or environmental tobacco smoke also increases the risk of respiratory infections and can result in a decrease in lung function. People with COPD experience a more rapid decline in what is called forced expiratory volume, or FEV1. However, if it does, the effect is small when compared to cigarette smoking. The use of solid fuels for cooking and heating may cause high levels of indoor air pollution, which may then lead to the development of COPD. Some patients who develop COPD have airway hyperresponsiveness, a condition in which their airways overreact to airborne irritants, such as secondhand smoke and air pollution. The role of airway hyperresponsiveness as a risk factor for COPD in people who smoke is unclear. Alpha1-antitrypsin (AAT) is a protein in the body that is produced by the liver and helps protect the lungs from damage. In AAT deficiency, the liver does not produce enough of this protein.

COPD is typically not diagnosed until the fifth decade of life. Common signs and symptoms of COPD are, a productive cough or an acute, breathlessness or being short of breath is the most significant symptom, but it does not usually occur until the sixth decade of life, wheezing is a musical, whistling, or hissing sound with breathing. Some people may wheeze, especially during exertion and when their condition worsens.

There is a considerable body of evidence that exercise training strategies in patients with chronic obstructive pulmonary disease (COPD) induce significant improvement in exercise tolerance and quality of life. Intensity and duration of exercise are important determinants of the physiologic adaptations that occur in response to training. In COPD patients there are indications that greater physiological benefits can be obtained through high-intensity compared to moderate-intensity training. However, high-intensity exercise training may not be applicable to those COPD patients who are unable to sustain such intensities for long periods of time due to symptom limitation. In fact patients with severe COPD are so limited by dyspnea and locomotor muscle weakness that their ability to exercise is restricted to very low-intensity levels.

Skeletal muscle dysfunction is another major factor that can contribute to exercise intolerance. This is evidenced by the findings that (1) forced expiratory volume in 1 s (FEV1) alone is a relatively poor correlate of exercise tolerance, (2) the perception of leg effort or discomfort is the main symptom that limits exercise in 40 to 45 percent of patients with COPD, and (3) exercise intolerance often persists after lung transplantation, when the patient's ventilatory limitation has been eliminated. Nutritional and psychological factors such as anxiety also frequently impact exercise performance. In addition to the above factors, the morphological and biochemical changes within the locomotor muscles of these patients including abnormal fiber-type distribution, reduced fiber cross-sectional areas decreased muscle capillarity and oxidative enzyme activities as well as mitochondrial dysfunction are associated with an early activation of anaerobic glycolysis, lactic acidosis and premature establishment of muscle fatigue during exercise

There have been different strategies employed to improve exercise capacity in patients with severe COPD, ie, heliox and oxygen supplementation, bronchodilator therapy, one-legged training, noninvasive mechanical ventilation (NIMV) as well as different training modalities of dis-continuous nature inducing lower ventilatory demands such as interval or intermittent exercise, upper limb exercise training and lower limb exercise training . Furthermore, in the rehabilitation setting strategies such heliox supplementation or NIMV would be expensive to implement in large cohort of patients whereas upper limb and lower limb exercise training could be cost effective and easily applicable

The amount of air which can be forcibly exhaled from the lungs in the first second of a forced exhalation. Measuring FEV1 is done through spirometry testing which helps your physiotherapist to determine your lung function. Because COPD causes the air in your lungs to be exhaled at a slower rate and in a smaller amounts compared to a normal, healthy person, measuring how well you can forcibly exhale air can help determine the presence of COPD. A decline in FEV causes a person to become short of breath and to have difficulty breathing.

Vital capacity (VC) is the maximum amount of air that can be inhaled or exhaled from the lung. It is one of the measurements taken during spirometry or pulmonary function testing. VC is measured using a spirometer. In COPD the vital capacity is reducing gradually.

The upper extremities play an important role in many activities of daily living such as bathing, dressing, hanging out the wash, and gardening. Patients with COPD frequently experience marked dyspnea and fatigue when performing these simple tasks. Upper limb activities

commonly require unsupported arm exercise, which poses a unique challenge for patients with COPD, whose upper limb muscles are required to act as accessory muscles of respiration. . During unsupported arm exercise, the participation of the accessory muscles in ventilation decreases, and there is a shift of respiratory work to the diaphragm .Upper limb exercise training included, throwing a ball against the wall with arms above horizontal in sitting position, passing a beanbag over the head in sitting position, exercises on overhead pulleys in sitting position ,moving a ring across a wire without touching the wire, while arm was above horizontal.

Lower-extremity endurance training is considered to be the cornerstone of pulmonary rehabilitation. A number of studies have shown positive results of this modality of training in pulmonary rehabilitation programs. . The lower-extremity exercise protocol was composed of 24 sessions of cycle training on a cycle ergometer . The patients performed three sessions per week, for 8 weeks. Initially, each session consisted of 30min of continuous cycling at HR peak obtained from the cycle ergonometric test

Conventional physiotherapy techniques like diaphragmatic breathing, pursed lip breathing exercises and chest mobilization technique are generally given to COPD Subjects. Pursed-lip breathing helps get rid of this air. Breathe in slowly through your nose for two seconds. Purse your lips like you are going to whistle. Breathe out through pursed lips for four seconds or more until your lungs feel empty. Breathe out naturally; don't push the air out of your lungs. Diaphragmatic breathing: This technique promotes diaphragm use and recruits the lower respiratory muscles. Breathe in slowly and deeply through your nose.

While breathing in, push your stomach out. Place your hand on your stomach so you can feel your stomach going out. Breathe out slowly and deeply through your mouth. While breathing out, let your stomach relax. Feel your stomach going in with your hand. Count 1, 2 when breathing in. Count 1, 2, 3, 4 while slowly breathing out.

Chest Mobilization Exercise: Make the patient in a comfortable setting position with hand rest on the thigh. Ask the patient to breathe in slowly and while inspire raise the upper limb in elbow exterior to 180° and hold the breath and upper limb to 5 sec. Ask the patient to expire slowly and during expiration bring the upper limb to starting position.

## **1.1 STATEMENT OF THE PROBLEM**

Statement of problem can defined as

‘Comparative analysis of upper limb and lower limb exercise training along with conventional physiotherapy in improving FEV1 and vital capacity in COPD subjects’

## **1.2 NEED OF STUDY**

- This study is aimed to find out effects of upper limb exercise training in improving FEV1 and vital capacity in COPD subjects.
- This study is aimed to find out effects of lower limb exercise training in improving FEV1 and vital capacity in COPD subjects.
- This study used to compare the upper limb and lower limb exercise training in improving FEV1 and vital capacity in COPD subjects.



## **1.3 HYPOTHESIS**

### **1.3.1 Null hypothesis**

There is no significant difference between upper limb and lower limb exercise training along with conventional physiotherapy in improving FEV1 and vital capacity in COPD subjects.

### **1.3.2 Alternative hypothesis**

There is significant difference between upper limb and lower limb exercise training along with conventional physiotherapy in improving FEV1 and vital capacity in COPD subjects.

## **1.4 OPERATIONAL DEFINITION**

### **Lower limb exercise training:**

Cycle ergometer training exercise done by the patient as instructed by the Physiotherapist.

### **Upper limb exercise training:**

Different type of exercises done by the patient by his upper limb overhead training in a specific pattern in sitting position.

### **Breathing exercise:**

A broad category of physical activity designed to increase strength and endurance of the respiratory muscles.

## **II. REVIEW OF LITERATURE**

### **2.1 SECTION A: LITERATURE REGARDING COPD**

**1. American Thoracic Society,(2004)** defined chronic obstructive pulmonary disease [COPD] is a preventable and treatable disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of lungs to noxious particles or gases, primarily caused by cigarette smoking. Although COPD affects lungs, it also produces significant systemic consequences.

**2.Satsharma et al., (2006)**

Defined COPD as a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema. The airflow obstruction generally is progressive, may be accompanied by airway hyper reactivity and may be partially reversible.

**3.WHO, (2007)**

Defined COPD as a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases.

**4.Hough A, (2005)**

Stated that a common disease entity of chronic bronchitis and emphysema is known as chronic obstructive pulmonary disease. COPD is a slowly progressive disease and most airways obstruction is fixed,

although some reversibility may be demonstrated with medication.

### **5.Cigna J .A et al.,(2005)**

defined Chronic obstructive pulmonary disease as a disorder of the pulmonary system characterized by limitations in airway flow rates, dyspnoea, coughing, abnormal sputum production and an inability to tolerate normal functional activities.

### **6.British Thoracic Society (1997)**

COPD is defined as chronic slowly progressive disorder characterized by airway obstruction( $FEV_1 < 80\%$  predicted &  $FEV_1/FVC$  ratio  $< 70\%$ ) which does not change markedly over several months. The impairment of lung function is largely fixed but is partially reversible by bronchodilator (or other) therapy

## **2.2 SECTION B :LITERATURE REGARDING INCIDENCE AND PREVALENCE OF COPD**

### **1.Fabbri L.M et al., (2005)**

conducted a study and that showed the prevalence of COPD determined by criteria of GOLD was 17.2 % among subjects older than 45 year. Prevalence increased with increasing age, especially in males, in those with more than 20 pack, years of smoking, and in low-income subjects.

### **2.Aggarwal A.N. et al., (2004)**

stated that the prevalence of COPD reported in different population studies from India is highly variable. The prevalence rates in male subjects of 2.12% studies reported from North are generally higher than 1.4% to 4.08% reported from South, respective range for female subjects vary from 1.3% to 4.9 % from North and from 2.55% to South India.

### **3.Thomson A, (1995)**

Commended that COPD is more common in middle to late adult life and in men more than women (ratio 5:1) .It is more common in urban areas than in rural areas.

### **4.S.K.Jindal et al; (2006)**

Population prevalence of COPD is very high in India with some centre to centre differences. COPD was diagnosed in 4.1% of 35295 subjects with a male to female ratio of 1.56:1 and a smoker to non smoker ratio of 2.65:1. Prevalence among bidi and cigarette smokers was 8.2% and 5.9% respectively. Odds ratio for COPD was higher for men, elderly individuals, lower socio economic status and urban residence.

### **5.V.Sobradillo et.al., (2000)**

In their study prevalence of COPD was 9.1%,15% in smokers ,12.8% in exsmokers and 4.1%in nonsmokers. Prevalence in men was 14.3% and 3.9% in women.

## **2.3 SECTION C: LITERATURE REGARDING ETIOLOGY OF COPD**

### **1.Turato. G et al., (2001)**

Stated that cigarette smoking is currently a casual factor of COPD in more than 90 % of patients, indicating that environmental factors are involved in the disease. The role of inherited alpha 1 antitrypsin deficiency has become well established as a risk factor for COPD.

### **2.Satsharma et al., (2006)**

Reported that the primary cause of COPD is exposure to tobacco smoke. Clinically significant COPD develops in15 % of cigarette smokers. Age of initiation of smoking, total pack-years, and current smoking status predict COPD mortality. Airway hyper responsiveness

stipulates that patient who has non-specific airway hyper reactivity and who smoke are at increased risk of developing COPD with an accelerated decline in lung function.

### **3.Scanlon P.D, (2004)**

Stated that the primary risk factor for COPD is tobacco use (predominantly cigarette smoking). Other factors are airway hyper responsiveness, exposure occupational dust and chemicals and genetic disorder causing alpha –1-antitrypsin deficiency.

### **4.Boggia, (2008)**

Reported that smoking habits and occupational exposure are confirmed as risk factors for COPD. Workers exposed to both risk factors have to be considered in COPD high risk class.

### **5.Jadwiga a (2002)**

COPD patients have elevated airway cytokine levels suggesting that the presence of increase in inflammation may increase their susceptibility to exacerbations. Rhinovirus infection is an important etiologic factor in COPD exacerbation.

## **2.4 SECTION D: LITERATURE REGARDING PATHOLOGY OF COPD**

**1.Celli B. R et al.,(2000)** stated that the basic pathophysiologic process in COPD consists of increased resistance to airflow, loss of elastic recoil, decreased expiratory flow rate and over inflation of lung. The hyper inflated lungs flatten the curvature of the diaphragm and enlarge the rib cage and these altered configuration of the chest activity places the respiratory muscle, including the diaphragm, at a mechanical

disadvantage and impairs their force generating capacity, metabolic work of breathing increased and increase the sensation of dyspnoea heightens.

**2.Starr J. A,(2001)** stated that in COPD chronic inflammation from inhaling pollutants causes hypertrophy of glands and goblet cells results in excessive mucus production, which either partially or completely obstruct the airway. Decrease in ciliary function also impair airway clearance and contribute to airway obstruction. When excessive secretions are present in an airway, air can be inspired around the secretion. During exhalation only small amount of air escapes before the airway close down around the secretion, trapping air distal to the mucus accounts for hyperinflation.

**3.Turato. G et al,(2001)** describes the major structural and cellular changes present in the peripheral airways, central airways and lung parenchyma of patients with COPD. The pathological hallmark of COPD is emphysema, bronchiolitis, and chronic bronchitis. Airflow limitations occur in emphysema by reducing the elastic recoil of the lung through parenchyma destruction, bronchiolitis by narrowing and obliterating the lumen and chronic bronchitis by mucus hypersecretion. The functional consequences of these abnormalities is expiratory airflow limitation. As flow is the result of a driving pressure and of an opposing resistance, it is refer to the changes in flow seen in smokers as airflow limitation, rather than airflow obstruction. Since both loss of elastic recoil and increase in airway resistance play an important role in decrease in flow.

**4.Russell M,(2000)** study suggests that smoking inadvertently damages the lining of the airway. As with any other part of the body in

response to injury, inflammation occurs. Inflammation stimulates the damaged lining to secrete mucus in an abnormal amount and also causes the airway to constrict.

**5. Shapiro S.D and Ingenito E.P, (2005)** stated that in COPD the pathological changes mainly affect the large airway, small airways and alveolar space. Large airway changes consist of mucus gland enlargement and goblet cell hyperplasia. The major site of increased resistance in most individuals with COPD is in airway 2mm or less. Characteristic cellular changes include goblet cell metaplasia and replacement of surfactant secreting Clara cells with mucus secreting and infiltrating mononuclear inflammatory cells. Smooth muscle hypertrophy may also be present. These abnormalities may cause luminal narrowing by excess mucus, oedema, and cellular infiltration. Fibrosis in the wall may cause airway narrowing directly and predispose to non-specific hyper reactivity. Changes in alveolar space are characterized by chronic inflammation and destruction with coalescence into larger alveolar spaces.

**6 .Henke O.M et al.,(2005)** commented that the anatomic disruption, ciliary impairment, plasma exudation and fibrin can change the viscoelasticity of the mucus and impair the surfactant properties of the airway lining material cause small airway obstruction and gas trapping. These changes in mucus clearance lead to ventilation-perfusion mismatch, impaired gas exchange, pulmonary hyperinflation, and inspiratory loading of the respiratory muscles leading to fatigue and ineffective cough.

## 2.5 SECTION E : LITERATURE REGARDING DIAGNOSIS AND INVESTIGATIONS OF COPD

1. **Watchie J, (1996)** stated that  $FEV_1$  is valuable in assessing the severity of airway obstruction.  $FEV_1 /FVC$  is the forced expiratory volume in one second expressed as a percentage of forced vital capacity. Normal  $FEV_1$  is  $\geq 75-80\%$  of predicted and  $FEV_1/FVC$  is 90%. In obstruction both  $FEV_1$  and  $FEV_1 /FVC$  is decreased.

2. **Bowes M,(2001)** suggested that the history is very important when considering a diagnosis of COPD and should include the patient's exposure to risk factors, smoking history and any respiratory or other medical conditions such as asthma or childhood respiratory tract infections. The most important test for verifying COPD is spirometry. Number of signs are evident upon physical examination including chest wall abnormalities such as barrel chest, hyperinflation of the lungs, low diaphragm position, diminished heart or breath sounds and rapid shallow respirations. The patient may also exhibit pursed lip breathing, the use of accessory respiratory muscles and retraction of intercostals spaces.

3. **Fabbri L.M et al., (2005)** stated that  $FEV_1$  remains the reference marker for diagnosis, assessment of severity and prognosis. Other lung function parameters may also be useful in assessing COPD. Low body weight in patients with COPD is associated with impaired pulmonary status, reduced diaphragmatic mass, low exercise capacity and higher mortality rate when compared with adequately nourished individuals with this disease.



**4. Allen M.B et al., (2005)** stated that the patient with COPD will have impairment of expiratory flow , best measured by spirometry. Blood gases should be performed to identify the severity of hypoxemia and decide if oxygen therapy is required. Chest X -ray is important, not for confirming the diagnosis of COPD but to exclude the other reasons for the presentation.

**5. Starr J.A, (2001)** reported that arterial blood gas analysis might reflect hypoxemia in the early stages of COPD. Hypercapnea appears as the disease progresses. With the disease progression, chest radiographs show several characteristic findings. These include depressed and flattened hemi diaphragms; alterations in pulmonary vascular markings; hyperinflation of the thorax evidenced by an increased antero-posterior diameter of the chest; and an increased retro sternal air space, hyperlucency, elongation of the heart and right ventricular hypertrophy.

## **2.6 SECTION F: LITERATURE REGARDING OUTCOME MEASURES OF COPD**

### **Pulmonary Function Test**

**1. Gildea T R, (2003)** stated that pulmonary function testing is a valuable tool for the evaluation of the respiratory system.

**2. Collins J.V, (1993)** suggested that simple measurements of FEV<sub>1</sub>, PEF<sub>R</sub> and FVC are useful for diagnosing airway obstruction. FEV<sub>1</sub>, PEF<sub>R</sub> and FVC will be reduced but the reduction in FEV<sub>1</sub> and PEF<sub>R</sub> is usually greater than FVC fall and FEV<sub>1</sub>/FVC ratio will be less..

**3. Pierce R., (2005)** stated that the degree of reversibility of an obstructive defect could be detected by measuring spirometry before and after administration of bronchodilator. An improvement in FEV<sub>1</sub> of 200ml or more infers significant reversibility, if the baseline FEV<sub>1</sub> is <1.5 L, as does an improvement of >15 % if the FEV<sub>1</sub> is > 1.5 L

**4.Sat Sharma et al., (2006)** stated that pulmonary function test are essential for the diagnosis and assessment of severity of disease, and they are helpful in following its progress. Lung volume measurements in COPD may document an increase in total lung capacity, functional residual capacity and residual volume and decrease in vital capacity .

**5. Celli B.R et al; (2000)** stated that the diagnosis of COPD should be considered in any patient who has the followings symptoms: cough; sputum production; or dyspnoea; or history of exposure to risk factor for the disease. Post bronchodilator spirometry is required to confirm the diagnosis of chronic obstructive lung disease. COPD presently is graded using a single measurement such as forced expiratory volume in one second.

**6.Allen M.B et al; (2005)** stated that the airflow limitation in COPD is best measured as forced expiratory volume in one second (FEV<sub>1</sub>) and this relates mortality. Lung function testing along with high-resolution thoracic computerized tomography (CT) scanning can result in a reasonably confident result.

## **2.7 SECTION G : LITERATURE REGARDING PHYSIOTHERAPY MANAGEMENT OF COPD**

**1.Faling J.L, (2002)** stated that controlled breathing techniques and chest physical therapy are the major component of rehabilitation of

patients with COPD. The goal of chest physical therapy is to facilitate the removal of excess secretions, thus reducing the resistance airflow and the work of breathing, improving pulmonary gas exchange.

**2.Pryor J.A et al., (2004)** commended that breathing control and relaxation positions improved the exercise tolerance in breathlessness patient. When the patient is sitting or standing, leaning forward, the abdominal contents raise the anterior aspect of the diaphragm and probably facilitating its contraction during inspiration.

**3. Cigna J A et al., (2005)** mentioned that the main components of chest physical therapy used to treat patients with COPD are : Positioning, Manual Techniques, Coughing Techniques, Breathing Techniques. Huffing avoids the dynamic airway collapse, bronchoconstriction and fatigue associated with uncontrolled forceful coughing.

**4.Massery M and Frownfelter D,(2003)** commended that patients with primary lung disease use their accessory muscles greatly increase the work of breathing secondary to the shortness of breath or coughing. Combination of relaxed diaphragmatic breathing and pursed lip breathing reduces the work of breathing in patients with COPD

**5. Hough A, (2005)** found that physiotherapy is often required to help clear secretions and reduce work of breathing including non-invasive ventilation to prevent intubation. Physiotherapy must include educating the patient and family about restoration and maintenance of exercise tolerance and basic self-management.

**6. Innocenti D M, Anderson J.M, (1993)** found that in chronic stage of COPD, chest shaking on expiration would assist in the removal of secretions. Effective huffing and coughing should be interspersed with

periods of relaxed diaphragmatic breathing. All patients should be taught diaphragmatic breathing and shown how to control breathing during attacks of dyspnoea. All breathless patients should be taught to control their breathing when walking on the floor or upstairs and hills.

**7.Kakizaki et al., (1999)** mentioned that thoracic mobility exercises reduced breathlessness and increased vital capacity in COPD patients.

## **2.8 SECTION H: LITERATURE REGARDING RESPIRATORY MUSCLES COPD**

**1.Crane L D , (1998)** mentioned that the sternocleidomastoid muscle moves the rib cage superiorly which expands the upper rib cage in the pump handle motion. Two important functions of the scalenes are that they counteract the downward pull of the parasternals on the sternum and expands the upper rib cage in its anterior posterior dimensions. The accessory muscles of inspiration assist in increase in thoracic diameters by moving rib cage up and out ward when the shoulder girdle is fixed.

**2.Ferguson (1993)** Lung Hyperinflation reduces the zone of apposition of the diaphragm. The net effect is decreased ability of the diaphragm to elevate the ribcage.

**3.Reid W D and Dechman G, (1995)** stated that during quiet inspiration in asymptomatic, the respiratory muscles contract in a coordinated fashion such that the diaphragm descends in a piston like fashion and the ribs move upward and outward. Activities such as exercise or even breathing at rest in individuals with respiratory diseases demand increased levels of ventilation which may require recruitment of both accessory inspiratory and expiratory muscles.

**4.Orozco-Levi M, (2003)** mentioned that respiratory muscles are essential for alveolar ventilation. These muscles work against increased mechanical loads due to airflow limitation and geometrical changes of the thorax derived from pulmonary hyperinflation in COPD patients.

**5.Lyn Hobson, (2003)** diaphragm is the principal muscle of respiration. It is estimated that two thirds of the vital capacity in all positions is contributed by diaphragm. In forced inspiration when the arms are fixed Pectoralis major draws the ribs toward the arms there by increasing thoracic diameter and pectoralis minor contract to elevate the ribs to which they are attached.

**6.Hough A, (2005)** found that COPD patients may lean forward on their elbows to force their diaphragm into a more efficient dome shape and stabilize the shoulder girdle for optimum accessory muscle action. Some patients can only inhale by lifting up their entire rib cage with their accessory muscles.

**7.Joanne Watchie, (1995 )**abdominals force diaphragm back to resting position and depress and compress lower thorax leading to increase in intra thoracic pressure which, which is essential for coughing.

**8.Sue Jenkins , (2004)** Normally expiration is passive, but with a decrease in expiratory airflow the patient may recruit the abdominal muscles and other expiratory and inspiratory muscles in an attempt to augment expiration

## **2.9 SECTION I : LITERATURE REGARDING UPPER LIMB AND LOWER LIMB EXERCISE TRAINING OF COPD**

**1.Carolyn L. Rochester, MD(2001)** Exercise training in chronic obstructive pulmonary disease the rationale for and outcomes of lower-

and upper-limb training, as well as ventilatory muscle training, are reviewed, and the potential for anabolic hormone supplementation to optimize the benefits of exercise training is discussed.

**2.F. Pitta<sup>a</sup>, A.F. Brunetto<sup>a</sup>,(1998)** Effects of Isolated Cycle Ergometer Training on Patients with Moderate-to-Severe Chronic Obstructive Pulmonary Disease.Despite the within-group changes, no between-group significant differences were observed. In COPD patients, the results of isolated low-to-moderate intensity cycle ergometer training are not comparable to effects of multimodality and high-intensity training programs.

**3.Janet I. Larson, margaret k.(1996)** Cycle Ergometer and Inspiratory Muscle Training in Chronic Obstructive Pulmonary Disease Home-based CET produced a physiological training effect and reduced exercise-related symptoms while IMT increased respiratory muscle strength and endurance. The combination of CET and IMT did not produce additional benefits in exercise performance and exercise- related symptoms. This is the first study to demonstrate a physiological training effect with home-based exercise training.

**4.V. S. Probst, T. Troosters (2001)** Cardiopulmonary stress during exercise training in patients with COPD. The cardiopulmonary stress resistance training is lower than during whole-body exercise and results in fewer symptoms. In addition, exercise testing based on guidelines using a fixed percentage of baseline peak performance and symptom scores achieves and sustains training intensities recommended according to the American College of Sports Medicine.

**5. Karla Gale, MS (1990)** Upper Arm Exercises Improve Vigor in COPD Patients, Unexpected was the improvement of general exercise capacity, the authors add, surpassing the minimal clinically important difference for patients with COPD.

**6.StefaniaCosti, Mauro Di Bari(2003)** Short-term Efficacy of Upper-Extremity Exercise Training in Patients With Chronic Airway Obstruction: A Systematic Review This systematic review shows that there is limited evidence examining UEET and that the evidence available is of poor quality. Therefore, a recommendation for the inclusion or exclusion of UEET in pulmonary rehabilitation programs for individuals with CAO is not possible. Further research is needed to definitively ascertain the effects of this training modality on patient-centered outcomes

**7.VillaPineta Hospital ,University of Modena and Reggio Emilia(2007)**

Efficacy of Arm Training in COPD Patients (UEET-COPD),Upper limb training for patients COPD improves upper limb exercise capacity, but has no additional effect on symptoms or quality of life, as compared with leg training alone.

**8.Subin,Vaishali Rao(2005)** Effect of upper limb, lower limb and combined training on health-related quality of life in COPD .The combined upper limb and lower limb training group showed a significant improvement in the exercise performance and health related quality of life.

**9.GanesanKathiresan(1995)** Review Article Effect of upper extremity exercise in people with COPD, This review suggests that in the

short term, arm endurance training improves arm exercise capacity and arm strength training improves arm strength. Further research is required, in people with COPD, to investigate the long-term effects of arm training.

**10. Anne E. Holland (2000)** Does Unsupported Upper Limb Exercise Training Improve Symptoms and Quality of Life for Patients With Chronic Obstructive Pulmonary Disease? Unsupported upper limb training for patients COPD improves upper limb exercise capacity, but has no additional effect on symptoms or quality of life, as compared with leg training alone. This type of upper limb training may not adequately address the complex interaction between respiratory mechanics and upper limb function.

**11 Carolyn Kisner Lynn Alen Colby, Therapeutic Exercise Foundation and Technique 3<sup>rd</sup> edition p664-665**

Breathing exercise are incorporate in into the overall pulmonary rehabilitation of program of patients with acute and chronic pulmonary disorder. Breathing exercise are designed to retain the muscle of respiration improve or redistribute ventilation, lessen the work of breathing, improve gaseous exchange ,oxygenation.

**12. Asha Hasimy Mohd Hasim, Dr Zainal Abidi, Muscle activity during diaphragmatic breathing compared to abdominal crunches – A pilot study**

Diaphragmatic breathing exercise are designed to improve the efficiency of ventilation ,decreased work of breathing. It is always used to mobilize the secretion during postural drainage.



### **13. Carolyn Kisner Lynn Alen Colby Therapeutic Exercise p671**

Diaphragmatic breathing is 3 dimensional involving all sides of lower ribs .It is done with the middle of torso involving a gentle expansion of lower rib as the diaphragm draws downward. This diaphragmatic breathing is marked by the expansion of abdomen rather than chest when breathing .it is the most efficient breathing compared to other techniques

### **14. Carolyn Kisner Lynn Alen Colby Therapeutic Exercise p671**

It is thought to be keep airways open by creating a back pressure in the airways. It is thought to help a patient with COPD studies suggest that purse lip breathing decreases respiratory rate and increases tidal volume and tolerance

### **15. Jennifer A Proyr Amnani Prasad Physiotherapy for respiratory condition 3<sup>rd</sup> edition**

Purse lip breathing is often used in patients severe airway disease. By opposing the lips during expiration the airway pressure inside the chest is maintained preventing the floppy airways from collapsing

### **16. E.H. Breslenn the pattern of respiratory muscle recruitment in purse lip breathing A chest journal**

Purse lip breathing is performed as expiratory blowing against pursed lip is a pulmonary rehabilitation strategy incentive or voluntary employed in patients with COPD to control dyspnoea. It provides apperception of control over breathing

### **3. METHODOLOGY**

#### **3.1 Study design**

Pre test and post test Comparative study

- Group A: lower limb exercise training group along with conventional physiotherapy
- Group B: upper limb exercise training group along with conventional physiotherapy

#### **3.2 Source of data**

MAGJ HOSPITAL, Mookkannoor, Ernakulam. Kerala

#### **3.3 Sample and Sampling method:**

20 subjects were selected and randomly assigned in to one of the two training groups

#### **3.4 Inclusion criteria**

- COPD above 45 years -60 years.
- Both male and female.
- Patients with chronic bronchitis.
- Patient had air flow obstruction. (FEV, FEC is <80%)

#### **3.5 Exclusion criteria:**

- Patients with unstable cardiac disease
- Acute rib fracture
- Subject not able to do PFT

- Patients with TB,
- Pneumonia
- Carcinoma of lung
- Psychiatric illness

### **3.6 Variables**

#### **Dependent Variables:**

1. FeV1
2. Vital capacity

#### **Independent Variables:**

1. Upper limb exercise training
2. Lower limb exercise training
3. Conventional physical therapy.

### **3.7 Tools and Materials:**

1. Pulmonary function test – using spirometer

### **3.8 Procedure**

The patient in Group A were instructed for upper limb exercise training along with conventional Physiotherapy.

The patients in Group B lower limb exercise training along with conventional Physiotherapy.

### **Treatment Procedure:**

The two groups are instructed to do exercise for once daily for eight weeks.

### **Upper limb exercise training**

Patients in this group had 10 minute of general warm up progressive upper limb training, and 10 minute of cool down

Upper limb exercise training in clued

1 throwing a ball against the wall with arms above horizontal in sitting position



Passing a beanbag over the head in sitting position. Exercises on over head pulleys in sitting position

Moving a ring across a wire without touching the wire, while arm was above horizontal

Duration: Each exercise was performed for 40 seconds followed by 20 seconds rest. Exercises would be repeated four times in four minutes. Minimum exercise training time is 30 minute .the patient un able to do 30 minute time, are progressively attain this duration at his 10th session

1. Warm up and cool down exercise for this section
2. Shoulder flexion extension
3. Shoulder abduction and adduction
4. Shoulder rotation
5. Elbow flexion and extension
6. Wrist flexion and extension
7. Exercise is performed for 5 times for both upper limbs

**Lower limb exercise training:**



The training program took place in the outpatient respiratory unit of the hospital . The lower-extremity exercise protocol was composed of

24 sessions of cycle training on a cycle ergometer. The patients performed three sessions per week, for 8 weeks. Initially, each session consisted of 30 min of continuous cycling at 80%  $HR_{peak}$  obtained from the cycle ergometer test. If the target of 30 min of continuous exercise was not possible to reach in the first sessions, the patients were allowed to cycle less time, keeping the intensity at 80% of  $HR_{peak}$ . In this case, the time of continuous cycling was gradually increased until the patient reached the 30-min target, which was achieved by all the patients until the 10th session. The increase in load during the 2 months was adapted according to the dyspnea ratings . After an initial period of 2 weeks , or until the patient was able to cycle continuously during 30 min the load was periodically adjusted to correspond to a dyspnea sensation scored by the patient as around 5–6 in a Borg scale . Any patient who showed  $SpO_2 < 90\%$  during exercise received oxygen by nasal canulae. In this case, the oxygen flow (1–3 liters/min) was adjusted as the minimum necessary to keep  $SpO_2 > 90\%$ .

### **Conventional physiotherapy:**

#### **Diaphragmatic breathing exercise:**

Prepare the patient in relaxed and comfortable position in reclined settling. Place therapist hand on rectus abdomen just below the anterior costal margin

Ask the patient to breathe slowly and deeply through nose. Have the patient keep shoulders relaxed and upper chest quiet allowing abdomen to rise

Then tell the patient to slowly at all air out using controlled expiration through mouth.

Practice this three or four limits and then rest do not allow the patient to hyperventilate, fore lift and prolonged expiration should be avoid.

### **Pursed Lip Breathing:**

Prepare the patient in relaxed and comfortable position such is half lying.

Explain the patient that expiration must be relaxed (passive) and that contraction of abdominals must be avoided

Place therapist hand over the patient's abdominal muscle to detect any contraction of abdominals. Instruct the patient to breathe in slowly and deeply then have the patient loosely purse the lips and exhale slowly twice as long inhalation patient is discouraged to perform forceful or prolonged expiation during the performance of the exercise.

### **Chest Mobilization Exercise:**

Make the patient in a comfortable setting position with hand rest on the thigh. Ask the patient to breathe in slowly and while inspire raise the upper limb in elbow exterior to 180\* and hold the breathe and upper limb to 5 sec.

The ask the patient to expire slowly and during expiration bring the upper limb to starting position.

## **Measurement procedure**

Pulmonary function testing is a method of determining how well lungs and airways are working. These tests may be done on a routine basis. It gives the Physiotherapist a series of numbers, which allows them to compare each patient's lung function with a predicted value based on age, height, and sex, as well as their lung function in the past.

The most common pulmonary function test is called a spirometry. During this test patient is asked to take in as deep a breath as possible, and blow out all of the air as fast and as hard as you can. Patients will make several attempts at this procedure to assure that the best performance has been measured.



## IV. DATA ANALYSIS & RESULT

### 4.1 Data Analysis

Paired' – test

The intra group analysis of results were done with Paired 't' test with 5% level of significance.

Statistical analysis is done by using dependent 't' test

$$t = \frac{\bar{d}\sqrt{n}}{s}$$

$$s = \sqrt{\frac{\sum d^2 - \frac{(\sum d)^2}{n}}{n-1}}$$

d = difference between the pre-test Vs. post test

$\bar{d}$  = mean difference

n= number of observations

s = standard deviation

To compare control Group and Experimental Group

Statistical analysis is done by using Unpaired 't' test

$$t = \frac{\bar{X}_1 - \bar{X}_2}{S} \sqrt{\frac{n_1 n_2}{(n_1 + n_2)}}$$

$$S = \sqrt{\frac{\sum d_1^2 + \sum d_2^2}{n_1 + n_2 - 2}}$$

Where

$S$  = Combined standard deviation

$d_1$  and  $d_2$  = Difference between initial and final readings in control group and experimental group respectively.

$n_1$  = No. of patients in control group

$n_2$  = No. of patients in experimental group

$\bar{X}_1$  and  $\bar{X}_2$  = Mean of control Group and experimental Group respectively.

**Table: 1**

S.NO	Variable FEV1	Improvement		Standard Deviation	Paired 't' value
		Mean	Mean Difference		
1.	Pretest	2.26	1.094	0.0364	94.88
2.	Posttest	3.36			

Table: 1 shows the pre & post test measurement value mean ,difference, standard deviation paired' t' value of Group A FEV1 values.

**Fig: 1**

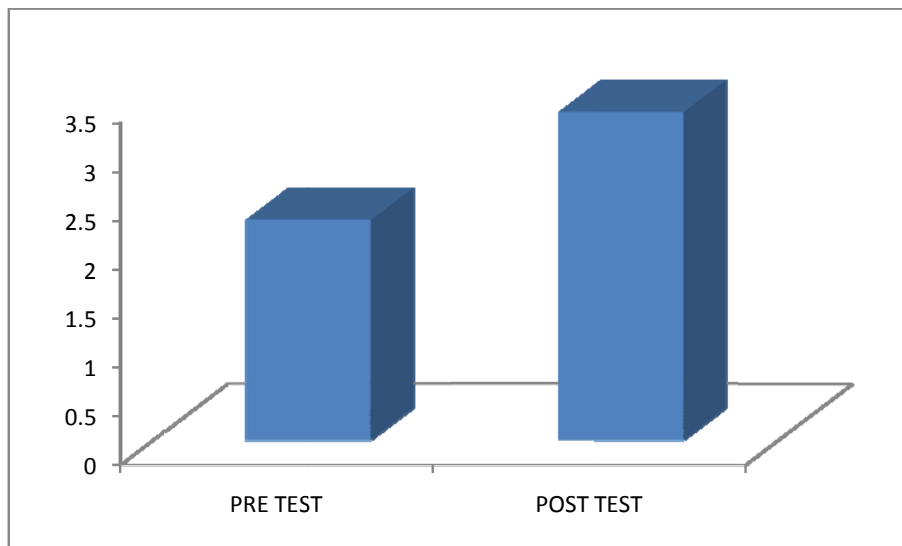


Fig: 1 shows the Group a pre & post test mean value of FEV1

**Table: 2**

S.NO	Variable FEV1	Improvement		Standard Deviation	Paired 't' value
		Mean	Mean Difference		
1.	Pretest	2.35	0.192	0.06766	8.978
2.	Posttest	2.54			

Table: 2 shows the pre & post test measurement value, mean difference, standard deviation paired 't' value of Group b FEV1 values

**Fig: 2**

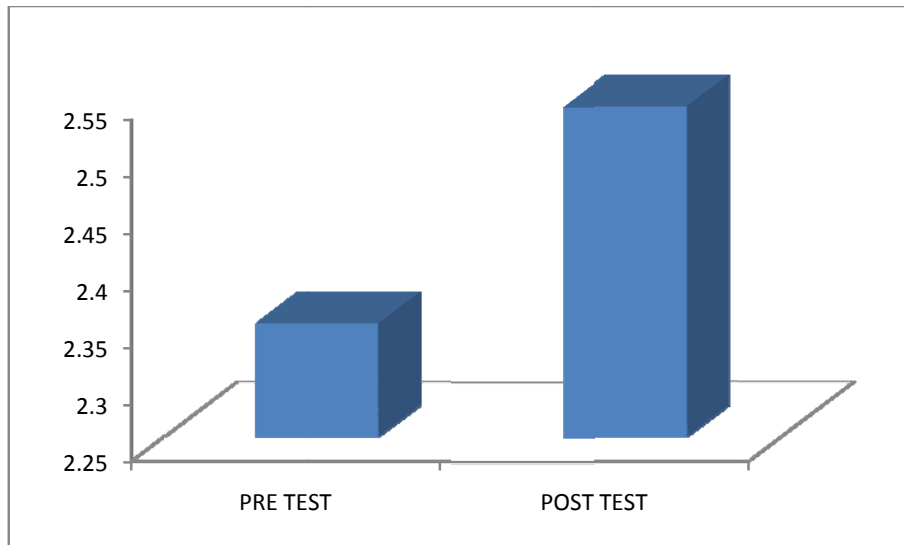


Fig: 2 shows the Group B pre & post test mean value of FEV1

**Table: 3**

S.NO	Variable vital capacity	Improvement		Standard Deviation	Paired 't' value
		Mean	Mean Difference		
1.	Pretest	3.18	0.82	0.1105	23.466
2.	Posttest	4.01			

Table: 3 shows the pre & post test measurement value mean, difference, standard deviation paired t value of Group A vital capacity values.

**Fig: 3**

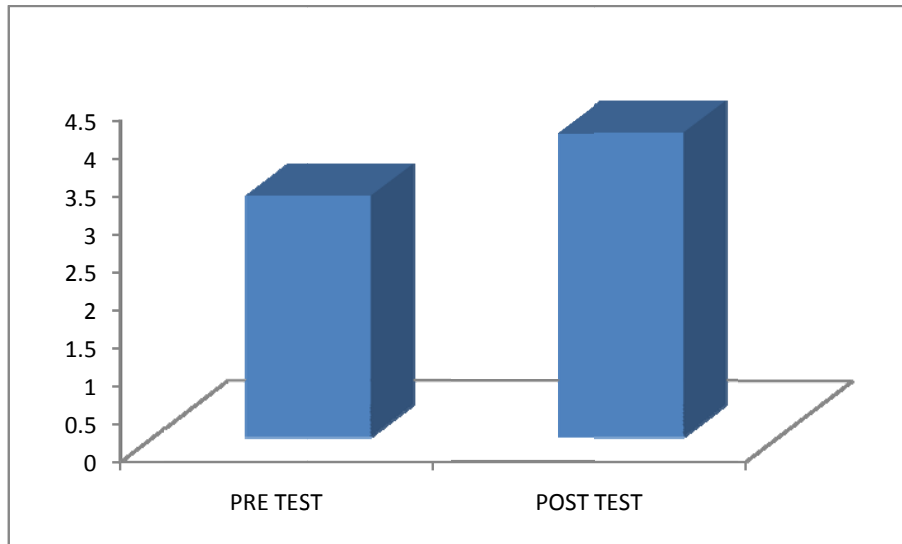


Fig: 3 shows the Group B pre & post test mean value of vital capacity

**Table: 4**

S.NO	Variable Vital capacity	Improvement		Standard Deviation	Paired 't' value
		Mean	Mean Difference		
1.	Pretest	3.4	0.207	0.0684	9.291
2.	Posttest	3.25			

Table: 4 shows the pre & post test measurement value, mean difference, standard deviation paired 't' value of Group B vital capacity values.

**Fig: 4**

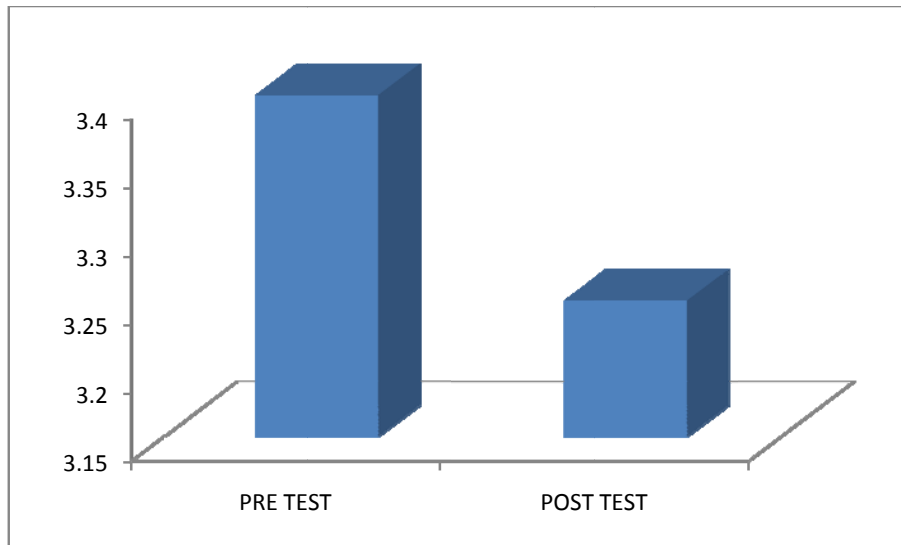


Fig: 4 shows the Group B pre & post test mean value of vital capacity

**Table: 5**

S.NO	Variable FEV1	Improvement		Un paired 't' Value
		Mean Difference	Standard deviation	
1.	Group A	1.094	0.08286	24.3471
2.	Group B	0.192		

Table : 5 shows the comparative mean difference, standard deviation and unpaired 't' value of FEV1

**Fig: 5**

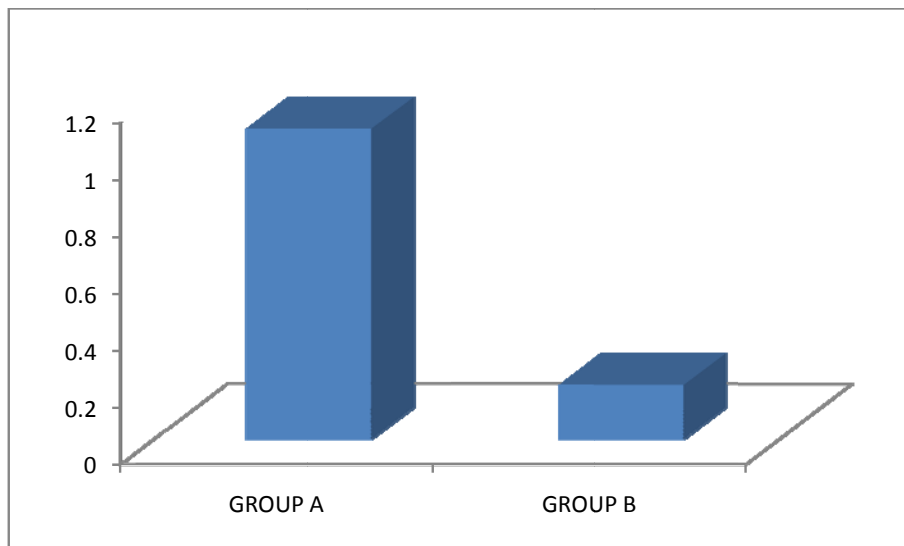


Fig: 5 shows the comparative mean difference value of Group A&B FEV1 data's

Table: 6

S.NO	Variable Vital capacity	Improvement		Un paired 't' Value
		Mean Difference	Standard deviation	
1.	Group A	0.82	0.05413	25.321
2.	Group B	0.2		

Table : 5 shows the comparative mean difference, standard deviation and unpaired 't' value of vital capacity

Fig: 6

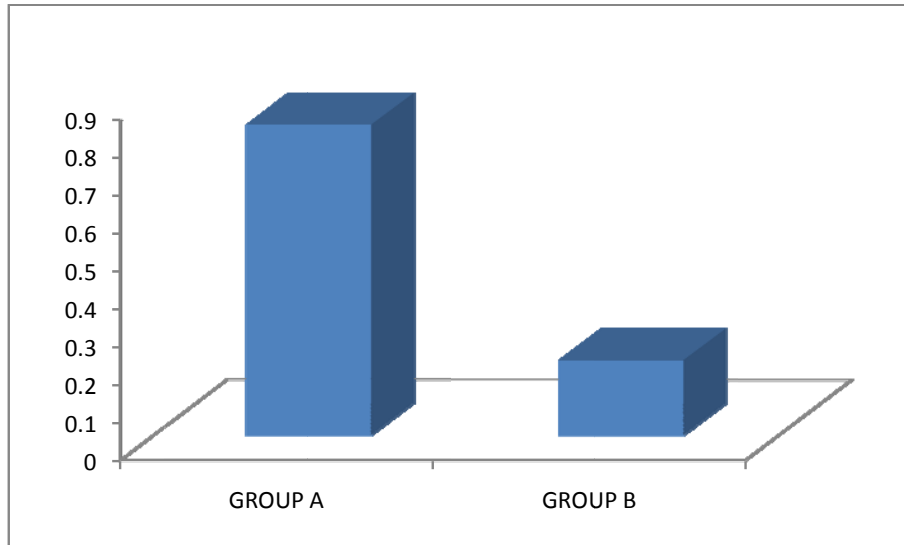


Fig: 6 shows the comparative mean difference value of Group A&B vital capacity data's



## 4.2 RESULTS

The number of subjects for the study were 20 (n=20). The subjects were divided in to two groups. For one group A (10 subjects) upper limb exercise training was given and for other group B (10 subjects) lower limb exercise training was given.

The subjects were given training for 2 months continuously. Before starting the training pretest value is measured. The measurement was repeated after the training (post test value). Reading pre and post test value of group A and B are given in table. Thus in both groups there was significant improvement scores of FEV1 and vital capacity.

When analyzing the fev1values of group A, by paired 't' test the calculated t value for is 94.88 and 't' table value is 4.78 at 0.001 level. Since the calculated 't' value is more than 't' table value, it is concluded that there is significant difference in the pre and post test values of FEV1 following upper limb training among COPD subjects.

When analyzing the FEV1 values of group B, by paired 't' test the calculated t value is 8.978 and 't' table value is 4.78 at 0.001 level. Since the calculated 't' value is more than 't' table value, it is concluded that there is significant difference in the pre and post test values of FEV1 following lower limb training among COPD subjects.

When comparing both group values of FEV1 by unpaired 't' test the calculated t value is 24.34 and 't' table value is 3.92 at 0.001 level. Here since the calculated 't' value is more than 't' table value, it is concluded that there is significant difference among upper limb training and lower limb training in improving FEV1 among COPD subjects.oke

patient. Hence the alternate hypothesis is accepted and null hypothesis is rejected.

When analyzing the vital capacity values of group A, by paired 't' test the calculated t value is 23.46 and 't' table value is 4.78 at 0.001 level. Since the calculated 't' value is more than 't' table value, it is concluded that there is significant difference in the pre and post test values of FEV1 following upper limb training among COPD subjects.

When analyzing the vital capacity values of group B, by paired 't' test the calculated t value is 9.291 and 't' table value is 4.78 at 0.001 level. Since the calculated 't' value is more than 't' table value, it is concluded that there is significant difference in the pre and post test values of FEV1 following lower limb training among COPD subjects.

When comparing both group values of vital capacity by unpaired 't' test the calculated t value is 25.32 and 't' table value is 3.92 at 0.001 level. Here since the calculated 't' value is more than 't' table value, it is concluded that there is significant difference among upper limb training and lower limb training in improving FEV1 among COPD subjects. oke patient. Hence the alternate hypothesis is accepted and null hypothesis is rejected.

## V. DISCUSSION

American Thoracic Society defined chronic obstructive pulmonary disease [COPD] is a preventable and treatable disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of lungs to noxious particles or gases, primarily caused by cigarette smoking. Although COPD affects lungs, it also produces significant systemic consequences.

In current study compare two groups , group A and group B. Group A getting upper limb exercise training along with conventional physiotherapy and group b getting lower limb exercise training along with conventional physiotherapy. Pre and post test done during the treatment period.

Efremidis G, Tsiamita M Accuracy of pulmonary function tests in predicted exercise capacity in COPD patients. They stated that conclude that exercise capacity was predicted from measurements of resting pulmonary function parameters with excellent accuracy in the COPD patient. . So present study included “Pulmonary function test “ for cardio pulmonary function.

Ries AL, Ellis B, Hawkins RW Upper extremity exercise training in chronic obstructive pulmonary disease. In this study shows improvement in the lung function according to his result.

L. LARSON JANET, Cycle Ergometer and Inspiratory Muscle Training in Chronic Obstructive Pulmonary Disease in this study the researcher state that lower limb training is beneficial in copd and it

increases the lung function .

Ries AL, Ellis B , Hawkins RW in their study shows that FEV1 and vital capacity are improved and also patients show improving there upper arm muscle power . The upper extremities play an important role in many activities of daily living such as bathing, dressing, hanging out the wash, and gardening. Patients with COPD frequently experience marked dyspnea and fatigue when performing these simple tasks. Upper limb activities commonly require unsupported arm exercise, which poses a unique challenge for patients with COPD, whose upper limb muscles are required to act as accessory muscles of respiration. This study shows improvement in cardiopulmonary.

L. LARSON JANET, Cycle ergometer training also improves the vital capacity and FEV1 in COPD patients. Walking and stair climbing are difficult to COPD patients and in daily living activity it is more important. During this study patient shows improvement in walking and other daily living activity. According to this study patient shows improvement in cardiopulmonary functions.

In this present study found that both upper limb and lower limb exercise training is beneficial to increase fev1 and vital capacity in COPD subjects .hence this study shows upper limb exercise training group shows more improvement in COPD subjects.

## **VI. CONCLUSION**

Chronic obstructive pulmonary disease defined as a usually progressive with air flow limitation that is not fully reversible and that is associated with abnormal inflammatory response of the lungs to noxious particles or gases. Physical therapy combining breathing exercise, chest Physiotherapy, are used. This study projects that both upper limb and lower limb training are effective in the treatment options in COPD.

When comparing the mean value of all the group, Group A which received upper limb training showed more difference than other two group which received lower limb training. Hence we can conclude that upper limb training are more effective than lower limb training in improving FeV1among COPD patients.

### **LIMITATIONS**

- Food habits, extremely cold food like, ice cream etc.
- Personal habits like smoking, alcoholism, tobacco
- Exposure to climate.
- The subjects do not have any follow up programme.
- Size of the sample was very small which might affect the outcome

### **RECOMMENDATION**

- To establish the efficiency of the treatment a large sample size study is required.

- For more valid result, a long term study must be carried out.
- Follow up programs can be included to assess the long term effects of treatment.
- Further study can be conducted to check the effects of these techniques on other respiratory conditions.
- Specific condition wise study can be conducted.
- Other pulmonary variable can be included.
- The study can be done with a large sample size.

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## VIII ANNEXURE

### ANNEXURE-1

#### **RESPIRATORY ASSESMENT FORM**

##### **Subjective assessment**

Name

Age

Sex

Occupation

Chief complaints

Present medical history

Past medical history

Personal history

History of allergens

History of immunization

Associated problems

Family history

Socio economic history

Psychological history

##### **Subjective evaluation of cardinal symptoms**

1.Pain

Pleuritic

Muscle

Skeletal

Neuralgic

Angina

2.Cough

A . Effectiveness

B. Variation

C. Productive/non productive

3 Sputum

A. Color

B. Constituency

C. Smell

D. Quantity

4.Dysnoea

1-sterenous activity

2-on ordinary activity

3- on < ordinary activity

4-at rest

5.Wheeze

Diurnal variations

Postural variations

Aggravating factors

**Objective assessment**

Vitals

Pulse rate

Respiratory rate

Blood pressure

Temperature

On observation

- A. level of consciousness
- B. built of patient
- C. posture
- D. head and face evaluation

color

Distress

Puffiness

- E. Neck

Usage of accessory muscles

Distention of veins

- F. Chest

Expansion

Unmoving chest

Deformity

Moving chest

Pattern of breathing rate depth rhythm

I:e ratio

Symmetry of movement

External of movement

G. Extremity evaluation

Clubbing

Edema

Cyanosis

Tremor

Skin changes

H. External appliances

**On palpation**

A. Tracheal shift

B. Tenderness

C. Chest expansion

Auxiliary level

Nipple level

Xiphi sternal level

D. Accessory muscle palpation

E. Tactile fremitus

F. Movement of diaphragm

**On Percussion**

Resonant

Hyper resonant

Hypo resonant

Dull

**On auscultation**

Normal sounds

Tracheal

Bronchial

Broncho vesicular

Vesicular

Voice sounds

Broncho phony

Ego phony

Whispering pectoriloquy

Added sounds

Wheeze

Crepitus

Pleural rub

Pericardial rub

Heart sounds

On examination

Chest expansion

Spirometry

Musculoskeletal assessment

Neuromuscular assessment

## ANEXXURE -II

### FeV1 SCORE

Table :7

Group A		Group B	
Pre test	Post test	Pretest	Post test
2.26	3.34	2.27	2.5
2.27	3.33	2.3	2.52
2.28	3.39	2.4	2.70
2.28	3.36	2.20	2.35
2.27	3.38	2.40	2.57
2.26	3.34	2.47	2.60
2.26	3.35	2.56	2.67
2.27	3.39	2.20	2.50
2.28	3.38	2.30	2.45
2.26	3.39	2.40	2.56

Table :7 shows pre and post test of FEV1



## VITAL CAPACITY SCORE

Table:8

Group A		Group B	
Pre test	Post test	Pretest	Post test
3.17	4.02	3.40	3.6
3.19	4.01	3.2	3.5
3.20	4.03	3.34	3.46
3.16	4.03	3.56	3.70
3.17	4.02	3.52	3.76
3.18	4.01	3.27	3.57
3.20	4.01	3.30	3.50
3.19	4.03	3.52	3.76
3.18	4.02	3.40	3.52
3.17	4.01	3.50	3.65

Table : 8 shows pre and post test value of Group A and Group B vital capacity data's

**ANEXXURE -III**

**PATIENT CONSENT FORM**

I ..... Voluntarily consent to participate in the research named on study “COMPARETIVE ANALYSIS OF UPPER AND LOWER LIMB EXERCISE TRAINING ALONG WITH CONVENTIONAL PHYSIOTHERAPY IN INMPROVING FeV1 AND VITAL CAPACITY IN COPD SUBJECTS “

**Signature of patient**

**Signature of researcher**