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To Compare the Effectiveness of Incentive Spirometer and Inspiratory Muscles Trainer in Patients with Chronic Obstructive Pulmonary Disease

Jaya Negi[1], Niraj Kumar[2], Nishu Sharma[3], Archana Chauhan[4], Shama Praveen[5]

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Abstract

Introduction: Chronic obstructive pulmonary disease is characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways to noxious particles or gases. Exacerbations and co morbidities contribute to the overall severity in individual patients [1]. In India, according to National Commission on Macroeconomics and Health background paper by Murthy et al., the annual treatment costs for COPD had been estimated to be greater than Rs. 35,000 crores in 2011 and Rs. 48,000 crores in 2016 [3]. COPD produces obstruction to the airflow which affects both the mechanical function and gas exchange of the lung. Respiratory muscles must work harder to overcome this resistance and therefore it leads to weakness of the respiratorymuscles. Drug therapy is the main treatment in patients with COPD which includes bronchodilators, mucolytics, appropriate antibiotics and corticosteroids. Following drug therapy, physical rehabilitation is the only management which reduce dyspnea [4]. Resistive Inspiratory Devices are hand-held devices of varying diameter. The resistance is increased by decreasing the diameter of the devices and resistance is decreased by increasing the diameter of the devices airway [7].

Aims and Objectives of the Study: To compare the effectiveness of Incentive Spirometer and Inspiratory muscles trainer on ventilatory muscle strength on patients with COPD.

Methods: Thirty subject male or female with COPD aged between 40-80 years were selected according to convenience (purposive) sampling based on the selection criteria. Subjects were are randomly assigned into two group of 15 subjects each namely experimental Group A and control Group B. Group A was treated with Inspiratory muscles trainer and Group B with Incentive spirometer for a duration of 4 weeks.

Discussion: In this study, efforts were made to compare the effects of Incentive Spirometer and Inspiratory muscles trainer devices as a treatment for improving ventilatory muscle strength in patients with mild to severe dyspnea in COPD. The study was done on randomized 30 COPD patients with mild to moderate dyspnea diagnosed by physician. The patients were randomly divided into 2 groups consisting of 15 subjects each. Group A was treated with Inspiratory muscles trainer and Group B with Incentive spirometer for a duration of 4 weeks. The results demonstrated that the patients treated with both the intervention were highly significant in improving ventilatory muscle strength and hence decreasing the exertional dyspnea. However statistically there was significant difference between the two groups.

Conclusion: This study provided evidence to support the use of Incentive Spirometer and Resistive Inspiratory Devices to improve ventilatory muscle strength in patients with mild to severe dyspnea in COPD. In conclusion, both the treatment programs are inspiratory muscles trainer is more effective the incentive spirometer in improving Inspiratory Capacity and reducing dyspnea which could be due to improvement in ventilatory muscle strength.

Keywords: Datacollectionsheet;Wristwatch;Timer; Incentive Spirometry withaccessories;Threshold inspiratory muscle training device (Philips Company); Modified Medical Research Council Dyspnea Scale (mMRC); Baseline Dyspnea Index (BDI) and Transition Dyspnea Index (TDI).

Author Affiliation: [1]Lecturer [2]Associate Professor [3]Assistant Professor [4]Assistant Professor [5]Lecturer, Shri Guru Ram Rai Institute of Medical & Health Sciences, Patel Nagar, Dehradun, Uttarakhand 248001, India.

Corresponding Author: Niraj Kumar, Associate Professor, Shri Guru Ram Rai Institute of Medical & Health Sciences, Patel Nagar, Dehradun, Uttarakhand 248001, India.

E-mail: drnirajkumar25@gmail.com

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Introduction

Chronic obstructive pulmonary disease is characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways to noxious particles or gases. Exacerbations and co morbidities contribute to the overall severity in individual patients [1]. As per WHO, Non communicable diseases refer to "Diseases that are chronic, life style related and usually progressive when not intervened". This holds true for COPD also as it is chronic, progressive and most of the risk factors are lifestyle related (smoking, biomass fuel exposure etc). Recently, the BOLD study conducted in Pune, Mumbai and Srinagar reported overall COPD prevalence estimates of 6.25%, 6.8% and 16.05%, respectively [2].

In India, according to National Commission on Macroeconomics and Health background paper by Murthy et al., the annual treatment costs for COPD had been estimated to be greater than Rs. 35,000 crores in 2011 and Rs. 48,000 crores in 2016 [3]. COPD produces obstruction to the airflow which affects both the mechanical function and gas exchange of the lung. Respiratory muscles must work harder to overcome this resistance and therefore it leads to weakness of the respiratorymuscles. Drug therapy is the main treatment in patients with COPD which includes bronchodilators, mucolytics, appropriate antibiotics and corticosteroids. Following drug therapy, physical rehabilitation is the only management which reduce dyspnea [4].

Patients with COPD present diverse degree of dyspnea and deterioration in exercise capacity in association with impaired cardio pulmonary function. Weakness and deconditioning of the respiratory muscles and peripheral muscles reduce exercise capacity and quality of life. Most commonly the functions of the inspiratory muscles are found to be impaired (decreased strength and endurance). Ventilatory Muscle Training (VMT) is an important component of the physical rehabilitation which improves the strength and endurance of the respiratory muscles. The different types of ventilatory muscle training includes Incentive Spirometry, Inspiratory resistance training with various Resistive Inspiratory Devices, and different breathing techniques for the relief of dyspnea [5].

The incentive spirometer is a device that encourages patients with visual and other positive feedback, to maximally inflate their lungs and sustain that inflation. It is a common mode of postoperative respiratory therapy and involves deep breathing facilitated by a simple mechanical device. Maximal lung inflation is thought to open collapsed alveoli and thereby prevent and resolve atelectasis. Incentive spirometer (IS) is the treatment technique which utilizes the incentive spirometer for respiratorytherapy [6].

Resistive Inspiratory Devices are hand-held devices of varying diameter. The resistance is increased by decreasing the diameter of the devices and resistance is decreased by increasing the diameter of the devices airway [7].

Incentive Spirometry and Resistive Inspiratory Devices are widely used to improve inspiratory muscle strength and to reduce dyspnea. These devices offer resistance while performing inspiration. Incentive Spirometer is a simple instrument which provides visual and auditory feed-back to the patient while performing inspiration, so that patient can achieve their preset goals. It encourages deep breathing and a sustained inspiration [8].

Objectives

Need of Study

To best of our knowledge there are several studies has been done on COPD but no studies has been done to compare the effectiveness of Incentive Spirometer (ICS) and Inspiratory muscles trainer (IMT) in patients with COPD.

Aims and Objectives of the Study

To compare the effectiveness of Incentive Spirometer and Inspiratory muscles trainer on ventilatory muscle strength on patients with COPD.

Hypothesis

Experimental Hypothesis

There is significant difference between the effect of incentive spirometery and inspiratory musclestrainer.

Null Hypothesis

There is no significant difference between the effect of incentive spirometery and inspiratory muscles trainer.

Review of Literature

Lung Anatomy

Each lung is conical in shape. It has:

(1) An apex at the upper end;

(2) A base resting on the diaphragm;

(3) Three borders, i.e. anterior, posterior and inferior; and

(4) Two surfaces, i.e. costal and medial. The medial surface is divided intovertebral and mediastinal parts.

Fissures and Lobes of the Lungs

The right lung is divided into 3 lobes (upper, middle and lower) by two fissures, oblique and horizontal. The left lung is divided into two lobes by the oblique fissure. The oblique fissure cuts into the whole thickness of the lung, except at the hilum.

Root of the Lung

Root of the lung is a short, broad pedicle which connects the medial surface of the lung to the mediastinum. It is formed by structures which either enter or come out of the lung at the hilum. The roots of the lungs lie opposite the bodies of the fifth, sixth and seventh thoracicvertebrae [9].

The Blood Vessels

The lungs have two blood supplies. The first arises from the right ventricle and carries deoxygenated blood via the pulmonary artery to the pulmonary capillaries, and thence the pulmonary vein back to the left atrium.

Bronchial Tree

The trachea divides at the level of the lower border of the fourth thoracic vertebra into two primary principal bronchi, one for each lung. The right principal bronchus is 2.5 cm long. It is shorter, wider and more in line with the trachea than the left principal bronchus. The left principal bronchus is 5 cm. It is 1 longer, narrower and more oblique than the right bronchus. Each principal bronchus enters the lung through the hilum, and divides into secondary lobar bronchi, 1 one for each lobe of the lungs. Thus there are three 1 lobar bronchi on the right side, and only two on the 1 left side.

Each lobar bronchus divides into tertiary or segmental bronchi, one for each broncho pulmonary segment; which are 10 on the right side and 10 on the left side. The segmental bronchi divide repeatedly to form very small branches called terminal bronchioles. Still smaller branches are called respiratory bronchioles. Each respiratory bronchiole aerates a small part of the lung known as a pulmonary unit.

The respiratory bronchiole ends in microscopic passages which are termed:

(i) Alveolarducts,

(ii) Atria,

(iii) Air saccules,and

(iv) Pulmonary alveoli. Gaseous exchanges take place in thealveoli.

Broncho pulmonary Segments

These are well-defined sectors of the lung, each one of which is aerated by a tertiary or segmental bronchus. Each segment is pyramidal in shape with its apex directed towards the root of the lung. There are 10 segments on the right side and 10 on the left. Inter segmental planes. Each segment is surrounded by connective tissue which is continuous on the surface with pulmonary pleura. Thus the broncho pulmonary segments are independent respiratory units [9].

Right lung		
Upper lobe	Middle lobe	Lower lobe
1. Apical 2. Posterior 3. Anterior	4. Lateral 5. Medial	6. Superior 7. Anterior basal 8. Medial basal 7. Lateral basal 8. Posterior basal

Left lung	
Upper lobe	Lower lobe
1. Apical 2. Posterior 3. Anterior 4. Superior lingular 5. Inferior lingular	6. Superior 7. Medial basal 8. Anterior basal 9. Lateral basal 10. Posterior basal

Physiology

A. The principal organs of the respiratory system include the nose, pharynx, larynx, trachea bronchi, and lungs. Within the lungs the main bronchi branch into 22 generations.

1. Air distribution to the gas exchangesurface.
2. Warming and humidifying theair.
3. Serving as a part of body defencesystem.
4. Preventing the alveolar oxygen and carbon dioxide partial pressures fromextreme changing

B. Air Flow and Airway Resistance

1. The volume of air that enters or leaves the alveoli per time unit is directly proportionate to the pressure difference and inversely proportionate to the airwayresistance.
2. The airway resistance is directly proportionate to the length of the airway and the magnitude of interactions between the flowing gas molecules, and it is inversely proportionate to r4 or r5 (r - airway radius).

3. When the breathing frequency is 15 times per minute, the airway resistance provides 28% of the total resistance toventilation.
4. Many factors, such as lung expansion, stimulation of muscarinic or beta-adrenergic receptors modify the airway diameter and, consequently, the airway resistance [10].

C. Gas exchange

Diffusion Gas exchange is the process of transferring gases across the alveolar and capillary membranes and it requires both diffusion of gas and perfusion of blood. Diffusion is a passive process, and it is for this reason that the lungs have evolved the structure that we see in terrestrial mammals.

Perfusion is such an important part of the gas exchange process that it merits specific consideration in relation to gas exchange Deoxygenated blood is returned to the lungs via the right side of the heart and the pulmonary artery. The latter is the only artery in the body to carry deoxygenated blood, which is distributed to a huge capillary network within the lung.

The factors influencing blood-flow distribution in the lungs include:

- Gravity (via alveolar pressure and hydrostaticpressure)
- Bloodvolume
- Cardiacoutput
- Pulmonary arterialpressure
- Pulmonary arterialresistance
- Lung volume (via alveolar pressure)
- Alveolar gas pressure (influenced by lung volume andgravity) [10].

Control of Breathing

Automatic control of the cardiovascular system, the respiratory system is under direct voluntary control, which is essential for a wide range of everyday activities, e.g., speaking, blowing, sniffing, straining, lifting, etc. The respiratory control center resides within the brainstem, receiving a myriad of inputs from somatic receptors, as well as from other parts of thebrain [11].

Mechanism of Respiration

Respiratory Movements

Respiration occurs in two phases namely inspiration and expiration. During inspiration, thoracic cage enlarges and lungs expand so that air enters the lungs easily. During expiration, the thoracic cage and lungs decrease in size and attain the pre inspiratory position so that air leaves the lungs easily. During normal quiet breathing, inspiration is the active process and expiration is the passive process [10].

Muscles of Respiration

Primary Muscles: The primary inspiratory muscles are the diaphragm and external intercostal. Relaxed normal expiration is a passive process. However there are a few muscles that help in forceful expiration and include the internal intercostal, intercostalisintimi, subcostals and the abdominal muscles.

Accessory Muscles: The accessory inspiratory muscles are the sternocleidomastoid, the scalenus anterior, medius, and posterior, the pectoralis major and minor, the inferior fibres of serratus anterior and latissimusdorsi, the serratus posterior anterior may help in inspiration also the iliocostaliscervicis. The accessory expiratory muscles are the abdominal muscles: rectus, abdominis, external oblique, internal oblique and transversusabdominis. And in the thoracolumbar region the lowest fibres of iliocostalis and longissimus, the serratus posterior inferior and quadratuslumborum.

Movements of Lungs

During inspiration, due to the enlargement of thoracic cage, the negative pressure is increased in the thoracic cavity. It causes expansion of the lungs. During expiration, the thoracic cavity decreases in size to the pre inspiratory position. Pressure in the thoracic cage also comes back to the pre inspiratory level. It compresses the lung tissues so that, the air is expelled out oflungs.

Authors Statements

Kisner *et al.* defined, COPD as obstruction of flow of air in the respiratory tract thus affecting ventilation and gas exchange. COPD are the disease of the respiratory tract that produce an obstruction to the airflow and that ultimately can affect both the mechanical function and gas exchanging capability of the lungs [17].

Hillegass EA, Sadowsky HS (2001) *et al*. Chronic bronchitis is defined as the hyper secretion of mucus, sufficient to produce a productive cough

on most days for 3 months during 2 consecutive years. Emphysema is abnormal and permanent enlargement of the air spaces distal to the terminal respiratory bronchiole, accompanied by destructive changes of the alveolar walls [18].

Donna Frownfelter, Elizabeth Dean (2006) *at el.* COPD is a disorder characterized by increase in airway resistance, particularly noticeable by prolonged forced expiration. Chronic bronchitis is a disease characterized by a cough producing sputum for at least 3 months and for 2 consecutive years [8].

Haslett C. Davidson S. Davidson's (1999) *et al.* COPD is chronic and slowly progressive disorder characterized by airflow obstruction (FEV1<80%) and chronic respiratoryfailure [19].

Sharma SK, Anand MP & Acharya VN (2003) *et al*. Various etiological factors are responsible for production of COPD. Cigarette smoking is one among the most prevalent risk factor for the development of COPD. As the tobacco exposure increases by hukka, bidi and cigarette, greater is the risk of developing COPD. Pipe and cigar smokers have higher morbidity and mortality from COPD than non- smokers although it is lower than cigarette smokers [20].

Sharma SK, Anand MP & Silverman (2003) *et al.* The cumulated amount of tobacco smoked is related to its adverse effects. Prolong smoking impairs the ciliary action and produces hypertrophy with hyperplasia of mucus secreting glands, further smoking inhibits the antiprotease and causes neutrophils to release proteolytic enzymes. Smoking causes recruitment of alveolar macrophages that's releases elastolytic enzymes and this elastase triggers emphysema [20,21].

O'Sullivan BS, Schmitz JT (2001) *et al*. Patients with COPD have airflow limitation due to airway obstruction. In emphysema exposure to chronic smoke leads to inflammatory cell recruitment within the terminal air space of the lungs. These inflammatory cells release elastolytic proteinase which damages the extra cellular matrix of lungs that leads to apoptosis of structural cells of the lungs. Inefficient repair of elastin and other extra cellular matrix component results in air space enlargement that defines pulmonary emphysema [23].

Gaude G S, Nadagouda & Katz MJ (2010-2011). In later stages of COPD, the patient does not have the energy to hyperventilate, so carbon dioxide builds up in the blood. Now the hypoxemia is accompanied by hyper- capnea (excess blood carbon dioxide), and the patient develops chronic respiratory acidosis, an ominous sign. Hypoxemia with acidosis is found in the late phase of the course of COPD [22,24].

Katz MJ (2010) *et al.* Chest x-rays are used to rule out other causes of airway obstruction, such as mechanical obstruction, tumours, infections, effusions, or interstitial lung diseases. In acute exacerbations of COPD, chest x-rays are used to look for pneumothorax, pneumonia, and atelectasis (collapse of part of a lung). When COPD includes significant chronic bronchitis, chest x-rays have a dirty look. There are more vascular markings and more nonspecific bronchial markings, and the walls of the bronchi look thicker than normal when viewed end on. Often, the heart appears enlarged [24].

Yoshimi K, Seyama K. Spirometry and Pitta F, Takaki (2007-2008) *et al.* Some of the other pulmonary function tests that are useful for understanding the pathophysiology of COPD include the diffusing capacity measurement of carbon monoxide per litter of alveolar volume (DLco/VA), measurement of lung volume using the nitrogen washout technique and whole body plethysmography, and measurement of lung compliance [25,26].

Methodology

Thirty (30) subjects were are randomly assigned into two group of 15 subjects each namely experimental Group A and control Group B. All the participant took a part in the experiments on a voluntary basis after signing a consent form and a demographic data was collected from each subject. This study was conducted in SMIH Hospital, Patel Nagar, Dehradun.

Inclusion Criteria: Age of 40-80 years of both sexes, Mild to moderate stable chronic obstructive pulmonary disease patients diagnosed by physician, Patient with an ability to perform incentive spirometry and inspiratory muscle training and Medically stable declared by the physician.

Exclusion Criteria: Patient with a history of asthma, allergic rhinitis or atopy are excluded.

Instrumentation: Data collection sheet, Wrist watch, Timer, Incentive Spirometry with accessories, Threshold inspiratory muscle training device (Philips Company), Modified Medical Research Council Dyspnea Scale (mMRC), Baseline Dyspnea Index (BDI) and Transition Dyspnea Index (TDI).

Procedure: Thirty (30) subjects were are randomly assigned into two group of 15 subjects each namely experimental Group A and control Group B. Group A was treated with Inspiratory muscles trainer and Group B with Incentive spirometer for a duration of 4 weeks.

Inspiratory Muscles Trainer Devices

The patients were positioned on treatment couch in semi Fowler's position with adequate back rest. Then patients were given a mouth piece of Resistive Inspiratory Device fitted with a specific aperture opening disc, and nose clip was placed on the nose, so that breathing was done through the mouth. They were instructed to inhale through the mouth piece of Resistive Inspiratory Device, which was instructed to keep in the mouth for the period of 1-minute. The training was gradually increased in such way that they were able to perform twice a day for 10 to 15 minutes in each session. The progression was initially focus on increasing the duration to 30 minutes, then the intensity was increased by using a smaller aperture disc [Fig. 1].

Fig. 1: Training with Inspiratory muscles trainer Devices

Incentive Spirometry

The patients were positioned on a treatment couch in semi-Fowler's position with adequate back support.

Patients were asked to take three to four slow, easy breaths and maximally exhale with the forth breath. The patients were asked to place mouth piece of Incentive Spirometer in mouth, and maximally inhaled through the mouth piece. As the patient inhaled through the mouth piece, a pressure drop occurs and causes the ball in the tube to rise to a level equivalent to the flow around it. At the end of maximal inspiration, the patients were asked to hold and then to exhale. This sequence was repeated for 10 to 15 times in each session. Treatment was given 2 times per day for the period of 4weeks [Fig. 2].

Data Analysis: The data was analyzed by Graph Pad Prism software version 8.0.1. Paired T-test used to compare (mMRC) modified medical research council dyspnea scale between experimental and control group. 2 Way Anova test used to compare to baseline dyspnea index (BDI) and transition dyspnea index between experimental and control group.

Fig. 2: patient performing Incentive spirometry

Results

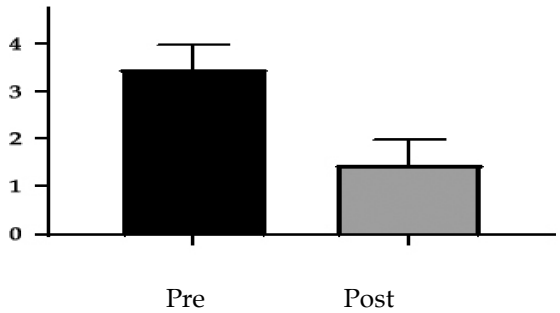
This results deals with the data analysis of the dyspnea scale between A and B. This course was analysed to compare the effectiveness of treatment protocols.

Paired T-test and 2 WAY- ANOVA test was used to compare the parameters of dyspnea between group A and group B.

Analysing mMRC revealed significant changes in pre-treatment experimental group with mean and SD (3.47 ± 0.5164) when compared with post treatment with mean and SD (1.47 ± 0.5164) [Table 1 & Graph 1].

Table 1: Mean and SD of mMRC of experimental group of pre and post treatment

Group	Mean ± SD	P value
Pre-treatment	3.47±0.5164	<0.0001
Post treatment	1.47±0.5164	<0.0001

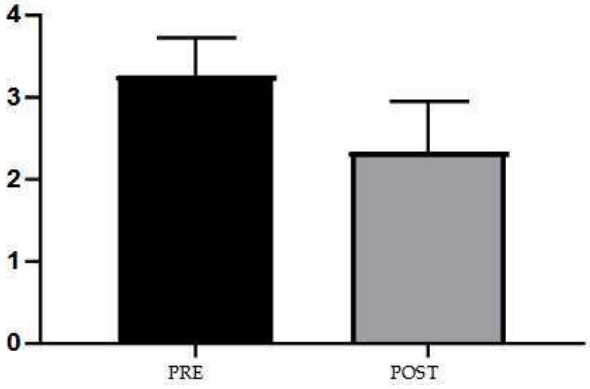


Graph 1: Comparison of Mean and SD of mMRC of experimental group of pre and post treatment

Analysing mMRC revealed significant changes in pre-treatment control group with mean and SD (3.27 ± 0.457) when compare with post treatment with mean and SD (2.33 ± 0.617) [Table 2 & Graph 2].

Table 2: Mean and SD of mMRC of control group of pre and post treatment

Group	Mean ± SD	P value
Pre treatment	3.27±0.457	<0.0001
Post treatment	2.33±0.617	<0.0001

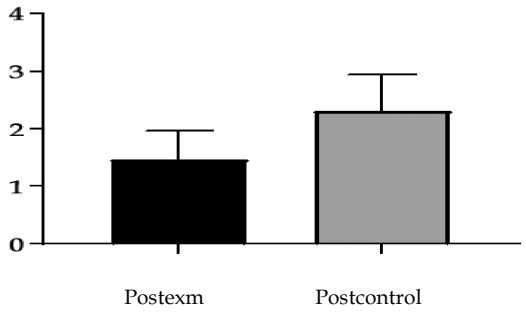


Graph 2: Comparison of Mean and SD of mMRC of experimental group of pre and post treatment.

Analysing mMRC revealed significant changes in experimental group with mean and SD (1.47 ± 0.5164) when compared with control group with mean and SD (2.33 ± 0.6172). [Table 3 & Graph 3].

Table 3: Mean and SD of mMRC of experimental group (group A) and control group (GroupB).

Group	Mean ± SD	P value
Experimental Group A	1.47±0.5164	<0.0001
Control Group B	2.34±0.6172	<0.0001

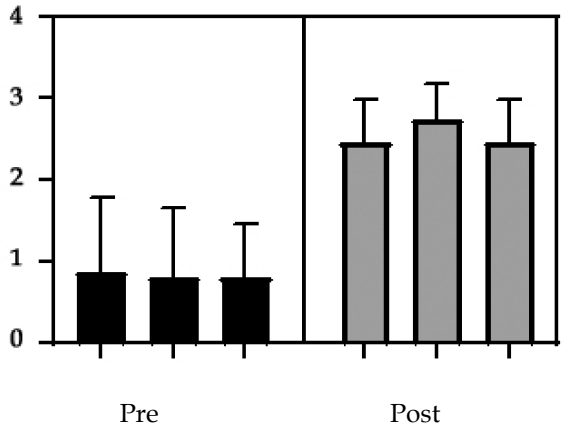


Graph 3: Comparison of Mean and SD of mMRC post treatment of experimental group and control group.

Analysing BDI and TDI revealed significant changes in pre-treatment experimental group with mean (3.47) when compared with post treatment with mean (1.47) [Table 4 & Graph 4].

Table 4: Mean and SEM of BDI/TDI of experimental group of pre and post treatment

Group	Mean± SEM	p value
Pre-treatment	0.83±0.09428	<0.0001
Post treatment	2.57±0.09428	<0.0001

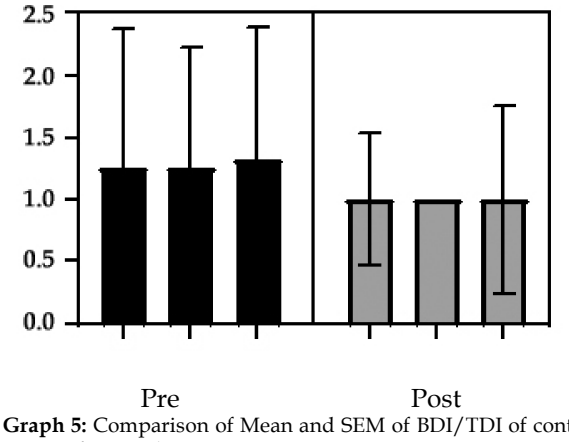


Graph 4: Comparison of Mean and SEM of BDI/TDI of experimental group of pre and post treatment

Analysing BDI and TDI revealed significant changes in pre-treatment control group with mean (1.29) when compared with post treatment with mean (1.00) [Table 5 & Graph 5].

Table 5: Mean and SEM of BDI/TDI of control group of pre and post treatment

Group	Mean± SEM	P value
Pre treatment	1.29±0.0948	<0.0033
Post treatment	1.00±0.0948	<0.0033

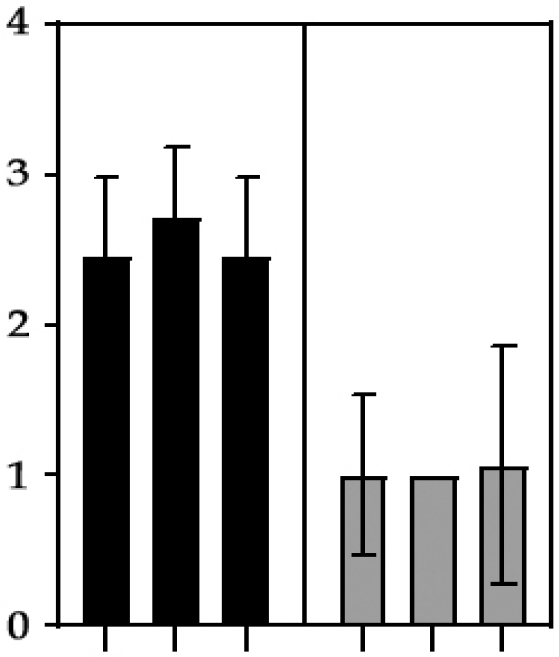


Graph 5: Comparison of Mean and SEM of BDI/TDI of control group of pre and post treatment

Analysing BDI and TDI revealed significant changes in experimental group with mean (2.56) when compared with control group with mean (1.02). [Table 6 & Graph 6].

Table 6: Mean and SEM of BDI/TDI of experimental group (group A) and control group (Group B)

Group	Mean ± SEM	p value
Experimental group	2.57± 0.1066	<0.0001
Control group	1.03± 0.1066	<0.0001



Graph 6: Comparison of Mean and SEM of BDI/TDI post treatment of experimental group and control group

Discussion

In this study, efforts were made to compare the effects of Incentive Spirometer and Inspiratory muscles trainer devices as a treatment for improving ventilatory muscle strength in patients with mild to severe dyspnea in COPD. The study was done on randomized 30 COPD patients with mild to moderate dyspnea diagnosed by physician. The patients were randomly divided into 2 groups consisting of 15 subjects each. Group A was treated with Inspiratory muscles trainer and Group B with Incentive spirometer for a duration of 4 weeks. The results demonstrated that the patients treated with both the intervention were highly significant in improving ventilatory muscle strength and hence decreasing the exertional dyspnea. However statistically there was significant difference between the 2 groups.

In the present study to aim to find the efficacy of which mode of treatment was better in the two group using two different evaluating tools such as mMRC, BDI/TDI. This scale is both reliable and valid significantly correlated with lung function and maximal exercise performance in patients significantly correlation between changes in maximal inspiratory pressure and commonest in the transitional dyspnea index support the concept increase inspiratory muscles strength may reduce dyspnea.

An improvement in inspiratory muscles strength and endurance might reduce symptoms and improve functional capacity in patients with severe COPD, even if airflow obstruction does not improve. Inspiratory muscles training is recommended for COPD patients and in a recent meta-analysis.

The "t" test and 2 way ANOVA was done to find out the significant of the data between two groups. Overall 15 COPD patient receive incentive spirometer technique and 15 patients receive inspiratory muscles trainer device technique. Who were selected based on the selection criteria. The results demonstrated that the patients treated with both the intervention were highly significant in improving ventilatory muscle strength and hence decreasing the exertional dyspnea. Based in this data we accept the experimental hypothesis and reject null Hypothesis. The study undertaken included patients who had COPD with mild to severe dyspnea.

In our study the mean flow of group A and group B varied between 2.57 and 1.05 I/s respectively.

We have shown that targeted inspiratory muscle straining result in significant increase in respiratory muscles functional and significant reduce in dyspnoea in clinically stable patients with mild to severe COPD.

Improvement occurs in both groups (these results may be due to treatment protocol which we have taken in this study). In this study proved that inspiratory muscles trainer is more effective than incentive spirometer in improving respiratory muscles strength and reduce dyspnea

The drawback of this study is and IS is simple device that can easily pursed and be used at the bed side of inspiratory muscle training and threshold device is very expensive and not easily available in themarket.

Conclusion

This study provided evidence to support the use of Incentive Spirometer and Resistive Inspiratory Devices to improve ventilatory muscle strength in patients with mild to severe dyspnea in COPD. In conclusion, both the treatment programs are inspiratory muscles trainer is more effective the incentive spirometer in improving Inspiratory Capacity and reducing dyspnea which could be due to improvement in ventilatory muscle strength.

Limitations of the Study

- The study is conducted for a short duration and

no follow up is done with the patients so, study shows only immediate effects and not the long -term effects.

- In this study, the effects of extrinsic factors such as administration of drugs like Bronchodilators, Beta blockers, Corticosteroids, etc. and intakes of caffeine in the diet are not considered while including patients in thestudy.

Scope for Further Study

- Further study can be done to check the combined effects of Incentive Spirometer and Resistive Inspiratory Devices.
- The exact mechanism behind the reduction of dyspnea following training and the relationship between the reduction of dyspnea and ventilatory muscle training can be studied in more detail.

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