

Effect of Inspiratory Muscle Training by Using Breather Device in Participants with Chronic Obstructive Pulmonary Disease (COPD)

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ABSTRACT

Background: Inspiratory muscle weakness is a clinical feature in patients with COPD. Due to hyperinflation, the shortened diaphragms generate lower force during contraction, contributing to dyspnea and reduced exercise tolerance. Due to pathological changes the strength of inspiratory muscle is reduced which leads to early fatigue and reduce in functional capacity. Therefore, inspiratory muscle training in COPD patients is designed to enhance respiratory muscle function and to reduce the severity of breathlessness and improve exercise tolerance.

Objective: To study the effect of the Breather device on inspiratory muscle strength and functional capacity by measuring Maximum Inspiratory Pressure (Pimax) and 6 Minute Walk Distance (6MWD).

Methodology: Thirty participants both male and female diagnosed with COPD were included in the study and were divided into two groups. Baseline assessment was done using Pimax and 6MWD. Participants in group A performed inspiratory muscle training by using breather device and diaphragmatic breathing and group B performed diaphragmatic breathing exercise twice a day for 2 weeks. Reassessment was done and data was compared.

Results: There was a highly significant ($p < 0.0001$) difference in Pimax between the pre ($57.66 \pm 7.76 \text{ cmH}_2\text{O}$) and post ($75.33 \pm 9.90 \text{ cmH}_2\text{O}$) intervention in group A with significant difference of $56 \pm 6.3 \text{ cmH}_2\text{O}$ and $60 \pm 7.07 \text{ cmH}_2\text{O}$ between the groups. Also there was significant ($p < 0.0001$) difference in 6MWD between the pre ($273.66 \pm 25.07 \text{ m}$) and post ($273.66 \pm 25.07 \text{ m}$) intervention in group A with the mean difference of 54.73 m and 35.4 m when compared between the two groups.

Conclusion: The use of breather device for inspiratory muscle training showed significant improvement in Pimax and 6MWD in participants with COPD.

Key words: COPD; Pimax; 6MWD; IMT; Breather device.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a leading cause of morbidity and mortality worldwide. According to World Health Organization (WHO); COPD is defined as a lung disease characterized by chronic obstruction of lung airflow that interferes with normal breathing

and is not fully reversible. In India, as in 2016, three out of five leading causes of mortalities constitute non-communicable diseases whereas COPD is the second biggest cause of death in India today. ^[1]

Multiple pathological mechanisms likely contribute to the development of COPD. Across the world, cigarette smoking

is the most commonly encountered risk factor for development of COPD leading to a higher prevalence of respiratory symptoms and lung function abnormalities. [2] The pathological hallmarks of COPD are inflammation of the peripheral airways and destruction of lung parenchyma leading to emphysema caused by airflow limitation that is usually progressive and is associated with an abnormal inflammatory response of the lungs to inhaled noxious particles or gases. [3] This inflammatory response results in release of free radicals, cytokines and proteases (proinflammatory enzymes) due to activation of neutrophils which cause mucus hypersecretion, loss of lung elasticity and also impairment of surrounding tissues. [4]

The airflow limitation leads to hyperinflation of the thorax which reduces the elastic recoiling of the lung. [5] Expiratory airflow limitation and loss of the lungs elastic recoil promotes air trapping with an increase in End-Expiratory Lung Volume (EELV), a decrease in Inspiratory Capacity (IC), and ultimately results in lung hyperinflation. [3] This adaptation reduces the ability of diaphragm to contract during inspiration, and thus limit its ability to generate inspiratory flow leading to dyspnea and exercise limitation. [6] Thus Individuals with COPD experience inspiratory muscle dysfunctions due to the combined effect of increased work of breathing, hyperinflation, malnutrition, hypoxemia, hypercapnea and possible use of corticosteroids resulting in decreased inspiratory muscle strength and endurance. [7]

For evaluating functional capacity of patients with COPD, the self-paced 6MWD can be used to assess the submaximal level of functional capacity. It is a simple test that requires a 100-ft hallway but no exercise equipment and participants can choose their own intensity of exercise and are allowed to stop and rest during the test. [8] Respiratory muscle weakness can be known by measuring Maximum Inspiratory Pressure (Pimax) and Maximum Expiratory Pressure (Pemax) by using hand held pressure manometer. The most common forms of

Respiratory Muscle Training (RMT) generally include both Inspiratory Muscle Training (IMT) and Expiratory Muscle Training (EMT) component to a various extent. [9]

Resistive loading requires individuals to inspire or expire via a variable-diameter orifice, whereby, for a given airflow, the smaller the orifice the greater the load achieved. [10] The Breather is a respiratory device that applies respiratory muscle training against a resistance by breathing through different sized orifices, thereby increasing respiratory muscle strength. [11] Among treatment modalities, breathing techniques are also applied to alleviate symptoms and improve respiratory physiology. One of the breathing techniques is Diaphragmatic Breathing (DB), also called abdominal or deep breathing that is done by contracting of diaphragm muscle which is dome-shape sheet of muscle located horizontally between thoracic cavity and abdominal cavity. [12] It aims to reduce dyspnea by increasing diaphragmatic excursion and simultaneously reducing accessory muscle use (which contributes greatly to work of breathing) and correcting abnormal chest wall movement. [13]

The inspiratory muscles can be specifically trained, with improvement of both muscle strength and endurance by using the breather device. Therefore, respiratory muscle training in COPD patients was a rational decision designed to enhance respiratory muscle function and to reduce the severity of breathlessness and improve exercise tolerance.

MATERIALS AND METHODS

Participants: Total thirty participants both male and female diagnosed with COPD were included in the study. Participants were screened according to the inclusion and exclusion criteria. Participants with age group of between 45-65, diagnosed with grade I and grade II COPD and willing to participate were selected for the study. Participants who were hemodynamically

unstable and with other cardiorespiratory and neurological diseases were excluded from the study. The study received approval from Institutional Ethical Committee of Dr. APJ Abdul Kalam College of Physiotherapy, Pravara Institute of Medical Sciences, Loni. Written informed consent was taken from all the participants selected for the study.

PROCEDURE:

The intervention was given two times in a day for 2 weeks. The session time was 15-20 min for each participant. Thirty participants were divide into 2 groups; group A (n=15) and group B (n=15). Participants in group A received respiratory muscle training through the Breather device along with diaphragmatic breathing. Diaphragmatic breathing was performed in semi fowler’s position with 5-6 repetitions for 5 sets. After performing diaphragmatic breathing, participants were asked to perform respiratory muscle training through breathe device beginning with the easiest setting by rotating the inhale and exhale dials at a non fatigable resistance. Participants were instructed to inhale deeply and forcefully for 2-3 seconds, slight pause, then exhale forcefully for 2-3 seconds for 10 repetition with 2 sets. Whereas participants in group B performed only diaphragmatic breathing with same repetition as in group A.

Outcome measures:

Participants were assessed for the baseline parameters which include maximal inspiratory pressure (Pimax) and 6MWD before and after intervention. For assessing Pimax, a hand-held pressure manometer was used. Whereas 6MWD was recorded by measuring the distance that the participants was able to walk in 6 min in 100ft hallway/ corridor.

DATA ANALYSIS AND RESULTS

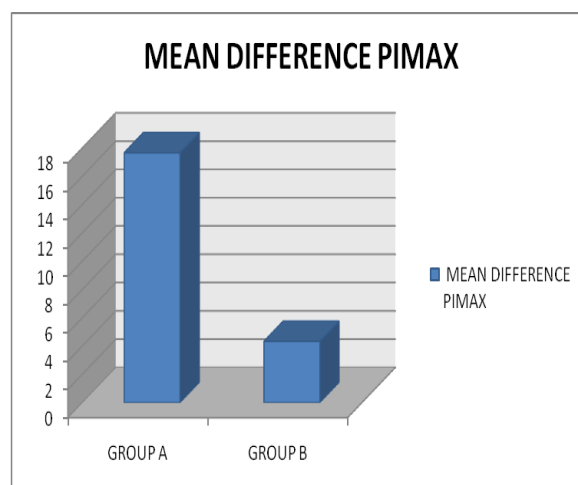
The data was entered into an excel spread sheet, tabulated and subjected to statistical analysis by using the trial version of Graph Pad Instat software. Descriptive statistics for all outcome measures were

expressed as mean, standard deviations and test of significance such as t test. The confidence interval was set at 95% and data was considered statistically significant with $p < 0.05$ and highly or considerably significant with $p < 0.001$.

Out of 30 participants, 15 participants were included in group A, in which 8 were male (53%) and 7 were female (47%) and 15 participants were included in the group B, in which 9 were male (60%) and 6 were female (40%). The differences in the baseline parameter for Pimax by participants in both the groups were significant ($p < 0.0013$, $t = 4.0$ with $df = 14$). The mean baseline value for Pimax in group A pre-intervention was 57.66 with SD of ± 7.76 and post-intervention was 75.33 with SD of ± 9.90 ($p < 0.0001$, $t = 12.909$ with $df = 14$) and in Group B pre-intervention was 56 with SD of ± 6.32 and post-intervention was 60 with SD of ± 7.07 ($p < 0.0013$, $t = 4.0$ with $df = 14$). The mean difference for Pimax in group A was 17.66 with standard deviation of ± 5.30 and in Group B was 4.33 with standard deviation of ± 3.20 ($p < 0.0001$, $t = 8.34$, $df = 28$) indicating highly significant.

Table 1.1:- Comparison of Pre-Intervention and Post-Intervention of Pimax in Group A and Group B

	MEAN \pm SD	Paired ‘t’ test value	‘p’ value
GROUP A	17.66 \pm 5.3	8.34	<0.0001 Highly Significant
GROUP B	4.33 \pm 3.2		

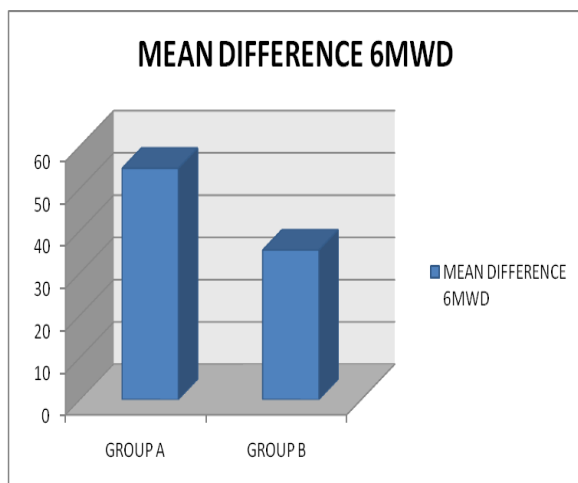


Graph 1.1:- Mean difference in Pimax in Group A and Group B

The differences in the baseline parameter for 6MWD by participants in both the groups were significant ($p < 0.0017$, $t = 3.47$ with $df = 28$). The mean difference for 6MWD in Group A was 54.73 with standard deviation of ± 4.43 and in Group B was 35.4 with standard deviation of ± 4.94 ($p < 0.0001$, $t = 11.28$, $df = 27$) indicating that there was highly significant difference in 6MWD in Group A when compared with Group B.

Table 1.2:-Comparison of Pre-Intervention and Post-Intervention 6 MWD in Group A & Group B

	MEAN \pm SD	Paired 't' test value	"p" value
GROUP A	54.73 \pm 4.43	11.28	<0.0001, Highly Significant
GROUP B	35.4 \pm 4.94		



Graph no.1.2 Mean Difference in 6 MWD in Group A & Group B

DISCUSSION

The main purpose of this study was to determine the effect of the Breather device on inspiratory muscle strength and 6MWD in participants with COPD when trained for 2 weeks. Total 30 participants were included in the study.

Hyperinflation is the pathological manifestation of airway obstruction, and the consequent expiratory flow limitation, which lead to incomplete lung emptying, i.e., expiration is reduced before the lungs have reached their equilibrium volume (FRC). [14] McKenzie DK et al, studied the Respiratory muscle function and its activation in chronic obstructive pulmonary disease in which they stated that chronically hyperinflated flattened state of the

diaphragm in COPD leads to shortening of the total diaphragm length by around 15% to 25%, depending upon whether it is assessed at functional residual capacity or residual volume respectively. This adaptation reduces the ability of diaphragm to shorten during contraction, and thus limit its ability to generate inspiratory flow. However, the adaptation in diaphragm structure and length appear to have some functional benefits in terms of maintaining its ability to deliver volume excursion, as well as its pressure-generating capacity. [15]

Ramírez-Sarmiento et al in their randomized control trail evaluated the structural changes in the respiratory muscles of COPD patients after a specific program of respiratory muscle training. They obtained sample of muscle fibres from external (inspiratory) intercostals muscle before and after 5 weeks of pressure threshold IMT. The training regimen consisted of 30 minutes training, 5 days per week at a load of 40-50% of MIP. Significant increases were observed in both the proportion of type I fibres (by 38%) and the size of type II fibres (by 21%) of the external intercostal muscles after the training period. These findings demonstrated that the external intercostal muscles of severe COPD patients have the capacity to express structural remodelling which leads to functional improvement induced by the inspiratory muscle training in terms of both inspiratory muscle strength and endurance that can be explained by structural adaptation within the inspiratory muscles. [16]

A study was conducted by Enright et al to examine whether high-intensity IMT resulted in changes in ventilatory function and exercise capacity in subjects who were healthy. Twenty subjects were randomly assigned to 2 groups, the training group completed an 8-week program of IMT set at 80% of maximal effort and the control group did not participate in any form of training. The training group demonstrated significant improvement in MIP (24% and 41%) after 4 and 8 week of training

respectively which clearly indicates that diaphragm hypertrophy is not only source of improvement in MIP, but it can also be improved through improvement in accessory muscle function as well as neural adaptations. These neural adaptations include an enhanced ability to coordinate the contraction of synergistic muscles, as well as to maximize the activation of individual muscles. [17]

Gosselink et al, in his study stated that diaphragm work is increased in COPD patients and they use a larger proportion of the Maximal Inspiratory Pressure (Pimax) than healthy subjects. This pattern of breathing is closely related to the dyspnea sensation during exercise which may induce respiratory muscle fatigue. Patients with a clear inspiratory muscle weakness and lower PaO₂ or higher PaCO₂ showed better response to IMT on inspiratory muscle strength and functional exercise capacity than those patients with better preserved respiratory muscle function. According to the force-length relationship, the higher the position of the diaphragm (the longer the resting length of the diaphragm, or the lower the lung volume), the higher the Pimax. [18]

Suh-Jen Lin et al, in their study stated that selective inspiratory muscle training is effective in patients with chronic heart failure. In patients with heart failure, Pimax lower than 70% of the predicted value indicates respiratory muscle weakness. Mechanisms underlying these beneficial effects of IMT included attenuated metaboreflex, improved ventilatory efficiency, and lower ventilatory oscillations during incremental exercise. Metabolic products accumulated from fatiguing respiratory muscle contraction could increase sympathetic vasoconstriction activity (the inspiratory muscle metaboreflex), and the attenuation of the metaboreflex could then improve blood flow redistribution to skeletal muscles in the body, thus delaying the time to fatigue and decreasing workload on the heart. [19]

The intercostal muscles and diaphragm contain specialized receptors

(muscle spindle) that respond to stretch. Muscle contraction stimulates a positive feedback loop via the spinal cord that increases motor drive to the inspiratory muscle. This response ensures that an increase in the resistance to inhalation is met with a compensatory increase in muscle recruitment. [5] The respiratory muscle also contain unmyelinated group III and group IV afferents that sense the metabolic state of the muscle, specifically the accumulation of metabolites such as lactate. These so-called metaboreceptors are present in all muscles and although they appear to have no role in the control of breathing they are important in the reflex control of cardiovascular system. [20]

Gandevia et al also stated that feedback signals from group III and group IV afferent has been implicated in central fatigue mechanism via inhibition of central motor output. It has been suggested that the afferent feedback from exercising muscle protects locomotor and respiratory muscle from catastrophic fatigue. An extreme example of central fatigue occurs with exercise of inspiratory muscle in which task failure can occur with minimal peripheral fatigue. [21] This could explain that weak inspiratory muscle can lead to early fatigue and so there was decrease in 6MWD in COPD patients in the present study. Therefore training respiratory muscle could somehow delay the central fatigue mechanism (delay in activation of metaboreflex) and increase the function capacity of patient.

Bailey et al, conducted a study to evaluate whether Inspiratory muscle training enhances pulmonary O₂ uptake kinetics and high-intensity exercise tolerance in humans. Sixteen active subjects were randomly assigned to receive 4 week of either pressure threshold IMT [30 breaths twice daily at 50% of maximum inspiratory pressure (MIP)] or sham treatment (60 breaths once daily at 15% of MIP). The subjects completed moderate, severe and maximal intensity "step" exercise transitions on a cycle ergometer before (Pre) and after

(Post) the 4-wk intervention period for determination of VO_2 kinetics and exercise tolerance. They found that there were no significant changes in the physiological variables of interest after sham treatment although after IMT, baseline MIP was significantly increased and the degree of inspiratory muscle fatigue was reduced after severe- and maximal intensity exercise. They stated that before IMT, breathing against an inspiratory load activates the inspiratory muscle metaboreflex, including vasoconstriction in both resting and exercising limb. After 4 week of IMT the metaboreflex response to the same loaded breathing task was delayed, and the time to fatigue during subsequent forearm exercise task was extended. The study also document a 54% increase in contracted diaphragm thickness, which explained 77% change in MIP. This study suggested that this mechanism (metaboreflex) plays an important role in mediating improvements in exercise tolerance. [22]

So in present study, improvement in MIP may leads to the delay in fatigue in the limbs which causes improvement in 6MWD in patients with COPD. The influence upon limb effort should now be clear, inspiratory muscle fatigue reduces limb blood flow, thereby reducing oxygen delivery and accelerating limb fatigue. Although it might seem self evident that weak or fatigued muscle generate greater perception of effort than fresh or stronger muscles.

In the present study, inspiratory muscle strength has significantly improved in the training groups who have received inspiratory muscle training through breather device. In addition participants in group A has shown significant improvement in 6MWD when compared to group B. This shows that inspiratory training have significantly improve the inspiratory muscle strength and functional capacity in patients with grade I and grade II COPD. Breathing against resistance during respiratory muscle training activates and strengthens respiratory muscle and improves quality of life by promoting regained activity and

exercise tolerance. This breather device functions as a therapeutic exerciser for the lungs and breathing muscle. It not only strengthens the respiratory muscle but also assists to breathe appropriately and efficiently. Furthermore, IMT adds significantly to general exercise regimes routinely recommended for patients with cardio-respiratory disorders. Compared to exercise alone, it has been reported that IMT is much more efficient in improving oxygen uptake and ventilation, maximal inspiratory pressure and exercise performance.

CONCLUSION

The present study showed that the use of breather device causes significant improvement in inspiratory muscle strength and 6MWD in experimental group when compared to the control group trained with only diaphragmatic breathing. Hence based on the above result we can conclude that addition of IMT in patients with COPD will increase the strength of inspiratory muscle as well as the functional capacity.

Limitation of Study:

- 1) The duration of the intervention was less because of shorter hospital stay of participants.
- 2) It was difficult to convince the participants to continue the treatment for longer duration and to keep their follow up after 2 weeks.
- 3) At times it was difficult for the participants to understand the instructions given to perform the breathing exercise through the breather device.

Suggestion for future research:

Future studies should be done for longer period of time and with larger sample size. Also IMT should be added to pulmonary rehabilitation to yield better result. For future studies, correlation of clinical data with long-term benefits such as hospitalization and readmission rates is also recommended.

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PIMS/CPT/IEC/2018/207

Conflict of Interest: None

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