Comparison of Intercostal Stretch Technique Versus Diaphragmatic Breathing on Dyspnoea, Chest Expansion And Functional Capacity in Stable Copd

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Abstract- Background: The use of diaphragmatic breathing and various proprioceptive neuromuscular facilitation (PNF) techniques has become more prevalent in cardiorespiratory physiotherapy to improve pulmonary functions. However, limited evidence exists regarding evaluation of their effectiveness in stable COPD. Hence, the objective of the study was to evaluate and compare the effectiveness of intercostal stretch technique versus diaphragmatic breathing on dyspnoea, chest expansion and functional capacity in stable COPD.

Methodology: Eighteen subjects were recruited based on inclusion and exclusion criteria. Dyspnoea score, chest expansion at three levels and 6 minute walking distance (6MWD) were obtained. Subjects were, then, randomly assigned to two groups. In group A, subjects underwent PNF technique using intercostal stretch for ten breaths in expiratory phase in supine lying position along with conventional chest physiotherapy, while in the group B, diaphragmatic breathing was given along with conventional chest physiotherapy.

Result: Intragroup analysis of dyspnoea score showed a very significant change in both groups (p=0.0039), 6MWD showed a very significant change (p=0.0028) in group A and a significant change in group B (p=0.0196), chest expansion changed non significantly at the axillary level (p=0.1690), extremely significantly at nipple level (p=0.0005) and at xiphisternal level (p<0.0001) in group A while it changed non significantly (p=0.0805) at the axilla level, very significantly (p=0.00081) at nipple level and extremely significantly (p<0.0001) at xiphisternal level in group B. Intergroup analysis of dyspnoea score, 6MWD and chest expansion at all three levels showed a non significant change.

Conclusion: Both intercostal stretch technique and diaphragmatic breathing are equally effective in reducing dyspnoea, improving chest expansion and functional capacity in stable COPD.

Index Terms: Diaphragmatic breathing, Dyspnoea, Chest expansion, COPD, Intercostal stretch

I. INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, primarily caused by cigarette smoking. COPD is the 4th leading cause of death worldwide and is expected to be the 3rd leading cause of death in the next 20 years.

The pathology of COPD includes inflammation and structural changes to all anatomical regions of the lung. Loss of bronchio-alveolar attachments leads to reduction of the elastic support normally given to the airways to maintain their patency, especially during expiration.

These abnormalities could all contribute to muscle weakness, particularly at sub-maximal activation, even in patients with mild to moderate COPD contributing to increased anatomical dead space in the lungs & dynamic hyperinflation that increase in end-expiratory lung volume (EELV) that may occur in patients with airflow limitation when minute ventilation increases. Obstruction causes ventilation perfusion mismatch resulting in hypercapnia, hypoxemia and increased oxygen demand leading to dyspnoea. Dyspnoea is the most common symptom of COPD.

The Medical Research Council (MRC) dyspnoea scale has been in use for many years for grading the effect of breathlessness on daily activities. The MRC dyspnoea scale is a simple and valid method of categorizing patients with COPD in terms of their disability.

Majority of studies dealing with factors contributing to inspiratory muscle weakness in COPD have focused on the diaphragm, mainly because the diaphragm is the principle muscle of inspiration. Patients with COPD have a lower transdiaphragmatic pressure generating lower capacity than healthy subjects, which has been described as hyperinflation-induced diaphragm shortening, placing the diaphragm at a mechanical disadvantage leading to reduced chest expansion.

Patients with COPD are markedly inactive in daily life due to dyspnoea and reduced chest expansion leading to a downward spiral of symptom-induced inactivity, causing deconditioning and muscle weakness which results in spending less and less time walking and standing compared with sedentary healthy elderly subjects leading to a reduction in functional capacity. Consequently, exercise testing is increasingly being used in the functional assessment of COPD patients.

A recent review of functional walking tests concluded that “the 6MWT is easy to administer, better tolerated and more...
reflective of activities of daily living than the other tests and it is practically simple. Diaphragmatic breathing is the normal mode of respiration. It moves the abdominal wall predominantly during inspiration to reduce upper rib cage motion which causes relaxation of the accessory muscles and thus decreases the work of breathing. Several studies have been done to assess the effectiveness of diaphragmatic breathing in COPD patients.

Proprioceptive Neuromuscular Facilitation (PNF) is a form of stretching in which a muscle is alternatingly stretched passively and contracted. The technique targets nerve receptors of a muscle to extend its length. It is usually a combination of passive stretching and isometric contraction. PNF is used to develop muscle strength, endurance, facilitate mobility, stability, control and coordinated movement and lays a foundation for the restoration of function. In addition, PNF also is known to improve lung functions. The facilitatory stimuli are intercostal stretch, vertebral pressure to the upper thoracic spine, vertebral pressure to the lower thoracic spine, anterior stretch lift to the posterior basal area, moderate manual pressure, perioral pressure, abdominal co-contraction. Intercostal stretch is effective in restoring normal breathing pattern, proving beneficial in improving the chest wall mobility and thus improving chest expansion. Vikram Mohan (2012) assessed the effect of intercostal stretch on pulmonary functions in healthy males. It showed an improved lung volume & pulmonary functions with intercostal stretch. PNF was compared with respiratory muscle resistance training on respiratory rate during weaning off phase of mechanical ventilation in another study by (Sharma Rajiv et al, 2010). It was seen that respiratory rate reduced significantly with PNF training.

Very few studies are done to evaluate the effects of intercostal stretch versus diaphragmatic breathing on dyspnoea, chest expansion & functional capacity in COPD patients according to our knowledge and hence the purpose of this study was to compare the effectiveness of intercostal stretch versus diaphragmatic breathing in stable COPD.

II. METHODOLOGY

18 Patients diagnosed with COPD were selected to participate in this Prospective Randomized Interventional Trial.

Inclusion Criteria:
1. Subjects with FEV1 between 50 - 80% according to GOLD criteria
2. Subjects with no acute exacerbation in the past 4 months
3. Both males and females between the age group of 50 - 70 years

Exclusion Criteria:
1. Haemodynamically unstable subjects and those in acute exacerbation of COPD
2. Subjects already performing chest physical therapy
3. Subjects unwilling to participate in the study

All subjects were between the age group of 55-65 years with a mean age of 61.555 ± 3.127 They were informed about the nature of the study and a written consent was taken. Ethical clearance was obtained from the local ethical committee before beginning with the study. Demographic data of the subjects was obtained. The dyspnoea score was obtained using the MRC dyspnoea scale, chest expansion was measured at the anterior axillary fold, nipple and the xiphisternal levels with a measure tape and six minute walking distance (6MWD) was calculated using six minute walk test.

Subjects were then, randomly divided through a draw system into group A and group B. Subjects in group A were given PNF in the form of intercostal stretch in supine position by applying pressure to the upper border of 3rd rib in a downward direction that will widen the intercostal space above it. The application of the stretch was timed with exhalation phase and the stretched position was then maintained as the patient continued to breathe in his usual manner. This technique was given from the 3rd rib to the 8th rib along with conventional chest physiotherapy twice a day, 5 days in a week for 4 weeks.

Subjects in group B were given diaphragmatic breathing in semifowler position and one hand was placed over the rectus abdominis just below the anterior costal margin. Subject was asked to breathe in slowly and deeply through the nose by keeping the shoulders relaxed and upper chest quiet, allowing the abdomen to rise slightly and then they were instructed to relax and exhale slowly through the mouth. The technique was also given along with conventional chest physiotherapy twice a day, 5 days in a week for 4 weeks. Dyspnoea score was obtained again at the end of 4 weeks using the MRC dyspnoea scale, chest expansion was measured again at the 3 levels mentioned above & 6 minute walking distance was calculated by the 6 minute walk test. Data obtained was then statistically analyzed.
FLOW CHART OF STUDY DESIGN

III. RESULTS

In the present study, we compared the PNF technique in the form of intercostal stretch versus diaphragmatic breathing on dyspnoea score, chest expansion at 3 levels & 6 minute walking distance in stable COPD patients.

Graph pad prism6 was used for statistical analysis in this study. To determine the statistical significance, p value was set as p < 0.05. All data presented as mean ± standard deviation. Baseline characteristics were determined for all the outcome measures.

Table 1: Baseline analysis of group A and B

<table>
<thead>
<tr>
<th>Parameters</th>
<th>GROUP A</th>
<th>GROUP B</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (years)</td>
<td>61.555 ± 3.127</td>
<td>61.111 ± 4.595</td>
<td></td>
</tr>
<tr>
<td>Number of males</td>
<td>6</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Number of females</td>
<td>3</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Mean duration of COPD (years)</td>
<td>7.000 ± 4.031</td>
<td>7.111 ± 3.951</td>
<td></td>
</tr>
<tr>
<td>Number of smokers</td>
<td>2</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Number of non-smokers</td>
<td>7</td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

Dyspnoea score was compared pre & post intervention in both groups using Wilcoxon signed rank test while the post intervention dyspnoea scores of both groups were compared using Mann Whitney U test.

Student t test was used to analyze six minute walking distance & chest expansion values both within groups and between groups.

The pre intervention score of dyspnoea on MRC scale (p value = 0.9592), six minute walking distance (p value = 0.7000) and the chest expansion at the axilla level (p value = 0.7513), nipple level (p value = 0.8581) and xiphisternal level (p value = 0.7513) between both the groups was not statistically significant which showed that both the groups were homogenous.

Table 2: Comparison of dyspnoea score, 6MWD and chest expansion at 3 levels pre and post intervention in group A

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Pre</th>
<th>Post</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnoea score</td>
<td>2.444 ± 0.5270</td>
<td>1.555 ± 0.5270</td>
<td>0.0039</td>
</tr>
<tr>
<td>6 MWD (metres)</td>
<td>501.111 ± 43.141</td>
<td>511.111 ± 42.557</td>
<td>0.0028</td>
</tr>
<tr>
<td>Chest expansion – axilla level (inches)</td>
<td>1.1333 ± 0.1500</td>
<td>1.1555 ± 0.1333</td>
<td>0.1690</td>
</tr>
<tr>
<td>Chest expansion – nipple level (inches)</td>
<td>1.0444 ± 0.1590</td>
<td>1.1555 ± 0.1424</td>
<td>0.0005</td>
</tr>
<tr>
<td>Chest expansion – xiphisternal level (inches)</td>
<td>0.8666 ± 0.1500</td>
<td>1.0444 ± 0.1130</td>
<td>&lt;</td>
</tr>
</tbody>
</table>

After 4 weeks of intervention, dyspnoea score showed a statistically very significant change (p value = 0.0039), 6MWD also showed a statistically very significant change (p value = 0.0028). However, chest expansion in group A at axillary level showed a non-significant change (p value = 0.1690), at the nipple level showed extremely significant change (p value = 0.0005) and at the xiphisternal level, also showed extremely significant change (p value < 0.0001) following 4 weeks of intervention.
Table 3: Comparison of dyspnoea score, 6MWD and chest expansion at 3 levels pre and post intervention in group B

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Pre</th>
<th>Post</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnoea score</td>
<td>2.444 ± 0.5270</td>
<td>1.666 ± 0.5000</td>
<td>0.0039</td>
</tr>
<tr>
<td>6 MWD (metres)</td>
<td>493.33 ± 40.927</td>
<td>503.33 ± 42.793</td>
<td>0.0196</td>
</tr>
<tr>
<td>Chest expansion – axilla level</td>
<td>1.1444 ± 0.1424</td>
<td>1.1777 ± 0.1302</td>
<td>0.0805</td>
</tr>
<tr>
<td>expansion – nipple level (inches)</td>
<td>1.0333 ± 0.1225</td>
<td>1.1111 ± 0.1167</td>
<td>0.0081</td>
</tr>
<tr>
<td>Chest expansion – xiphisternal</td>
<td>0.9111 ± 0.1833</td>
<td>1.0444 ± 0.1667</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>level (inches)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

After 4 weeks of intervention, dyspnoea score showed a statistically very significant change in (p value = 0.0039), 6MWD also showed a statistically significant change (p value = 0.0196). Chest expansion, when measured in group B after 4 weeks of intervention, showed a non-significant change (p value = 0.0805) at the axillary level, a very significant change (p value = 0.0081) at the nipple level and showed an extremely significant change (p value < 0.0001) at the xiphisternal level.

Table 4: Comparison of dyspnoea score, 6MWD and chest expansion at 3 levels post intervention between groups A & B

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Group A</th>
<th>Group B</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnoea score</td>
<td>1.555 ± 0.5270</td>
<td>1.666 ± 0.5000</td>
<td>0.6762</td>
</tr>
<tr>
<td>6 MWD (metres)</td>
<td>511.11 ± 42.557</td>
<td>503.33 ± 42.793</td>
<td>0.7041</td>
</tr>
<tr>
<td>Chest expansion – axilla level</td>
<td>1.1555 ± 0.1333</td>
<td>1.1777 ± 0.1302</td>
<td>0.7252</td>
</tr>
<tr>
<td>expansion – nipple level (inches)</td>
<td>1.1555 ± 0.1424</td>
<td>1.1111 ± 0.1167</td>
<td>0.4793</td>
</tr>
<tr>
<td>Chest expansion – xiphisternal</td>
<td>1.0444 ± 0.1130</td>
<td>1.0444 ± 0.1667</td>
<td>&gt;0.9999</td>
</tr>
<tr>
<td>level (inches)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Dyspnoea score between both the groups after 4 weeks of intervention showed a non-significant change (p value = 0.6762) 6MWD between both the groups showed a non-significant change (p value = 0.7041) after 4 weeks of intervention.

Chest expansion at the axillary level (p value = 0.7252), nipple level (p value = 0.4793) and xiphisternal level (p value > 0.9999) also showed a non-significant change after 4 weeks of intervention between both the groups.

IV. DISCUSSION

COPD is a lung disease defined by persistently poor airflow as a result of breakdown of lung tissue and dysfunction of the small airways usually followed by dyspnoea, fatigue, and sputum production.15

Abnormalities in small airways and destruction of lung parenchyma contribute to the development of airflow limitation with a consequent reduction of lung elastic recoil leading to dynamic hyperinflation and thus shortness of breath.

Intercostal muscles help in upward and outward movement of the ribs which results in increase in the anterior posterior diameter of the thoracic cavity. It helps both in inspiration and forced expiration.13,16 It is seen that due to dyspnoea, the physical activities in COPD patients are reduced. This will have an impact on the oxidative capacity of the skeletal muscles and it will reduce the proportion of muscle fibers from type I to type II. Hence, it can be hypothesized that intercostal muscles which aids in the mechanical aspects of breathing may undergo atrophy when there is a poor physical activity. Therefore, this could have an impact on chest wall mobility and chest expansion in COPD leading to dyspnoea.13

Chest expansion reduces due to decrease in the chest wall mobility and reduced lung compliance. Intercostal stretch may enhance the chest wall elevation and thus increase expansion to improve intra-thoracic lung volume which contributes to improvement in flow rate percentage. This may contribute to the increase in ventilatory capacity such as tidal volume, minute ventilation and oxygen status, thus improving the chest expansion, hyperinflation and air trapping, in turn reducing dyspnoea. The changes in ventilatory parameters may be due to the firing discharge of the muscle spindle during a passive stretch phase.13,17 Intercostal stretching may have activated the stretch receptors in the chest wall, thereby distending the thorax which could be neurologically linked to medulla with efferent nerve cells.13

Diaphragm is the primary muscle of ventilation accounting for approximately 70% to 80% of the inspiration during quiet breathing. The thoracoabdominal movement during quiet inspiration is a result of the pressures that are generated by the contraction of the diaphragm, the shape of the diaphragm and angle of pull of its fibres. In COPD, the costal fibres of diaphragm become more horizontally aligned due to hyperinflation so that the further contraction of diaphragm no longer lifts the lower rib cage in a bucket handle movement. This leads to paradoxical breathing in which the lower rib cage is pulled inwards during inspiration.18 Thus, there is an alteration in the mechanical efficiency of diaphragm in COPD.

During diaphragmatic breathing, the abdomen becomes the fulcrum and lifts the lower rib cage and rotate it outwards, reducing dynamic hyperinflation of the rib cage and improving gas exchange, thus optimizing the pattern of thoraco abdominal motion.18,19 The idea behind decreasing dynamic hyperinflation of the rib cage is that this intervention will presumably result in diaphragm working over a more advantageous part of their length-tension relationship.20
Moreover, diaphragmatic breathing is expected to decrease the elastic work of breathing, because the chest wall moves over a more favourable part of its pressure volume curve. This may be responsible for reducing the work load on the inspiratory muscles along with the sensation of dyspnoea.\textsuperscript{20} Since the diaphragm has its major attachment at the xiphisternum, it could be the probable reason for a significant improvement of chest expansion at the xiphistermal level in this study.

With intercostal stretching and diaphragmatic breathing, the lung compliance and the chest wall mobility is improved. This may be the reason for an improved pulmonary ventilation thus, indirectly increasing the functional capacity.

V. LIMITATIONS

A possible limitation with this study is that the sample size is small. Also the duration of intervention is only 4 weeks. Another limitation is that a long - term follow up was not done. Hence the long – term beneficial effects of both intercostal stretch and diaphragmatic breathing exercises are not clearly known.

Hence, it can be recommended that a similar study can be carried out with a larger sample size and with a longer duration of intervention. Also a follow up after a period of 3 to 6 months may be done to ascertain the long – term effects of both the techniques in COPD patients.

VI. CONCLUSION

Thus, it can be concluded from the present study, that both PNF technique in the form of intercostal stretch and diaphragmatic breathing are equally effective in reducing dyspnoea, improving chest expansion and increasing the functional capacity in stable COPD. Hence, both the techniques can be incorporated in the rehabilitation program for stable COPD patients. However, intercostal stretch can be performed only by a therapist while diaphragmatic breathing exercises can be given as a part of a home program as it is easy for the patient to understand and learn and can be done daily without supervision.

REFERENCES


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