Benefits of Respiratory Muscle Stretch Gymnastics in Chronic Respiratory Disease

Minehiko Yamada1,2, Masato Shibuya1, Arata Kanamaru1, Kazumasa Tanaka2, Hajime Suzuki2, Murray D. Altose3 and Ikuo Homma1

Abstract: In-phase vibration, applied to the contracting chest wall respiratory muscles, has been shown to reduce dyspnea in patients with chronic obstructive pulmonary disease (COPD). In the present study, respiratory muscle stretch gymnastics (RMSG) were developed to stretch the chest wall respiratory muscles during the contraction phase. Thirty-four patients with COPD consecutively performed four RMSG patterns, four times each. The dyspnea rating on a 150-mm visual analog scale, was 11.6±3.4 mm before RMSG and was significantly decreased to 6.2±2.2 mm (p<0.01) 5 minutes after RMSG. Arterial oxygen saturation, systolic and diastolic blood pressures, and pulse rate remained unchanged. Forced vital capacity and peak expiratory flow rate were significantly increased from 1807±141 ml to 1923±145 ml (p<0.01) and from 2.03±0.27 L/sec to 2.26±0.27 L/sec (p<0.05), respectively. Other spirometric values remained unchanged. Our results suggest that RMSG is a safe and effective physical conditioning method to improve pulmonary function and to decrease dyspnea at rest in patients with COPD.

Key words: dyspnea, lung diseases, obstructive, rehabilitation

Introduction

Dyspnea, or the unpleasant sensation related to breathing, is often a major factor limiting activity of daily living and quality of life in patients with respiratory disease1. However, in cases of chronic obstructive pulmonary disease (COPD), the control of dyspnea remains difficult. Physical conditioning programs have long been considered important in the management of chronic lung disease. Various types of exercise2,3 and respiratory muscle training4,5 have been proposed. Recently, these conditioning programs have come into wide use as part of comprehensive rehabilitation programs6,7. Although physical conditioning increases exercise capacity and respiratory muscle endurance, methods are not standardized and the effect on dyspnea is still unclear5,8-12.

Afferent activity from chest wall respiratory muscles, presumably intercostal muscle spindles, are thought to be involved in the mechanism of dyspnea13. In-phase vibration (IPV), which increases the afferent activity from contracting intercostal muscle spindles, decreases dyspnea induced by hypercapnia and inspiratory resistive loading in normal sub-

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jects\textsuperscript{14}). Furthermore, IPV decreases dyspnea at rest in patients with chronic respiratory
disease\textsuperscript{15). This finding suggests that afferent activity from muscle spindles in the contract-
ing intercostal muscles decreases dyspnea. Muscle spindles are also stimulated by muscle stretching\textsuperscript{16}). If this stretch stimulation is applied to the contracting respiratory muscles, the afferent activity generated would be similar to that elicited by IPV. Likewise, this may decrease dyspnea.

In the present study, we developed an original program, termed respiratory muscle stretch gymnastics (RMSG), which is designed to stretch inspiratory muscles during inspiration and expiratory muscles during expiration. This stretching program was designed to be easy to learn, understand, and perform. The purpose of this study was to examine the effect of RMSG on dyspnea in patients with COPD and to evaluate the safety of the stretch program.

**Method**

**Subjects**

The subjects were 34 patients (33 men and 1 woman; age, 68.0±1.6 years; range, 46 to 85 years) with COPD who were hospitalized or treated as outpatients at Showa University Fujigaoka Hospital Department of Respiratory Medicine (Table 1). The clinical diagnosis of COPD satisfied the criteria of the American Thoracic Society\textsuperscript{17). Eleven patients were receiving home oxygen therapy. All study patients met the following entry criteria: 1) clinical diagnosis confirmed by history, physical examination, pulmonary function testing, and chest x-ray; 2) stable condition while receiving standard medical treatment before the study; 3) no unstable cardiac disease or other significant disease; and 4) gave informed consent but were unfamiliar with the effect of RMSG on dyspnea.

Before the study, all patients underwent pulmonary function testing, including spirometry and lung volume determinations (CHESTAC 55V, CHEST Co., Tokyo, Japan). Testing and quality control followed standard and recommended procedures\textsuperscript{18,19). The arterial blood gas tension on room air was measured with a gas analyzer (ABL330, Radiometer, Copenhagen, Denmark). Forced vital capacity (FVC) was 69.8±2.9% of predicted value. Forced expiratory volume in 1 second (FEV\textsubscript{1}) was 0.95±0.09 L. Functional residual capacity was 4.0±0.2 L. The ratio of residual volume to total lung capacity was 57.6±1.8%. The \( \text{PaO}_2 \) was 71.1±1.6 torr, and the \( \text{PaCO}_2 \) was 43.8±1.0 torr (Table 1).

<table>
<thead>
<tr>
<th>Table 1. Profile of the COPD patients.</th>
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<tbody>
<tr>
<td><strong>No. of patients</strong></td>
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<tr>
<td>Age (yr)</td>
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<tr>
<td>Sex (male : female)</td>
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<tr>
<td>Breathlessness</td>
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<tr>
<td>Dyspnea at rest on 150-mm VAS</td>
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<tr>
<td>Home oxygen therapy</td>
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<tr>
<td>(+ : −)</td>
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<tr>
<td>Pulmonary function data</td>
</tr>
<tr>
<td>%VC (%pred.)</td>
</tr>
<tr>
<td>FEV\textsubscript{1} (L)</td>
</tr>
<tr>
<td>FRC (L)</td>
</tr>
<tr>
<td>RV/TLC (%)</td>
</tr>
<tr>
<td>Resting arterial blood gas (room air)</td>
</tr>
<tr>
<td>PaO\textsubscript{2} (torr)</td>
</tr>
<tr>
<td>PaCO\textsubscript{2} (torr)</td>
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* mean±SE. VC, vital capacity; FEV\textsubscript{1}, forced expiratory volume in 1 second; FRC, functional residual capacity; RV, residual volume; TLC, total lung capacity; \( \text{PaO}_2 \), arterial oxygen pressure; \( \text{PaCO}_2 \), arterial carbon dioxide pressure.
Respiratory Muscle Stretching

Fig. 1. Illustration of respiratory muscle stretch gymnastics. See Methods section for a detailed description of the movement.

**RMSG**

RMSG was designed to stretch predominantly inspiratory muscles during inspiration and expiratory muscles during expiration. The upper chest wall intercostal muscles and back muscles consist mainly of inspiratory muscles, and the lower chest wall muscles consist mainly of expiratory muscles\(^{20-22}\).

The following four patterns of RMSG were performed (Fig. 1):

**RMSG No. 1:** From a relaxed position with a straight back, slowly elevate both shoulders while moving them backwards. At the same time, lean back while inhaling (left). After full inspiration, exhale slowly and resume original position (right).

**RMSG No. 2:** With the back straight, hold both hands at the back of the buttocks (left). After full and slow inspiration, push the hands away from the body while slowly exhaling (right). After full expiration, breathe quietly and resume original position.

**RMSG No. 3:** With the back straight, hold both hands in front of the chest with the fingers entwined and the palms in (left). Inspire fully in this position. Then extend the arms and bend the upper body as far forward as possible while exhaling slowly (right). After arm extension and body bending, take a full breath in that position. Then breathe quietly and resume original position.

**RMSG No. 4:** With the back straight, hold both hands above the head with arms stretched and palms facing down (left). After full inspiration in this position pull the arms back while
exhaling slowly (right). After full expiration, resume original position and breathe quietly.

**Protocol**

The four RMSG were demonstrated in front of each patient to insure good understanding of the movements. Patients were then asked to rest while sitting in a chair for 5 minutes. Next, blood pressure (BP) was measured. Pulse rate (PR) and arterial blood oxygen saturation (SaO₂) were measured with a digital pulse oximeter (552-P, CSI Co., Milwaukee, WI, USA). Next, the intensity of dyspnea at rest was measured using a 150-mm visual analog scale (VAS). Dyspnea was explained as the discomfort or the effort related to breathing, and the patients were instructed not to include other sensations when quantifying dyspnea by VAS. Spirometry was performed according to the standard method. Then the patients were asked to perform each RMSG four times in the order of No. 1, No. 2, No. 3, No. 4, and No. 1. Neither the speed nor the duration of movement was specified. The RMSG were performed either standing or sitting, depending on the patient's condition.

The SaO₂, PR, and BP were measured immediately after the complete RMSG regimen was performed. Then dyspnea at rest was quantified again after the patient had rested in a seated position for 5 minutes after RMSG. Results of the baseline VAS rating were not shown to the patients. Spirometry was repeated in the same manner. The patients performed RMSG while breathing room air. Oxygen was not administered for at least 30 minutes before RMSG and was not administered during the study in all patients. The protocol was approved by the Showa University Ethics Committee.

**Statistical Analyses**

Descriptive statistics were calculated for measurements of VAS, spirometry, BP, PR, and SaO₂ before and after RMSG. The VAS measurements were compared by the Wilcoxon single rank test, and spirometric values, BP, PR, and SaO₂ were compared by paired t-test. All data are expressed as mean±SE.

**Results**

**Effect on the intensity of dyspnea at rest (Fig. 2)**

Nineteen of 34 patients had dyspnea at rest (VAS value > 0). Fourteen of the 19 patients with dyspnea at rest had lower VAS values 5 minutes after performing RMSG than before performing RMSG. The VAS value did not change in 4 of the 19 patients, and was increased in 1 of the 19 patients. RMSG did not induce dyspnea in any of the 15 patients who were not dyspneic at rest (VAS value = 0). The VAS value of all 34 patients was significantly decreased from 11.6±3.4 mm to 6.2±2.2 mm (p<0.01). The VAS values of the 19 patients with dyspnea at rest (VAS value > 0) was also significantly decreased from 20.7±5.3 mm to 11.1±3.6 mm (p<0.01).

**Effect on blood pressure, pulse rate, and arterial oxygen saturation (Table 2)**

There was no significant change in systolic and diastolic blood pressure and SaO₂ before and after RMSG. There was a small increase in PR from the baseline value of 85.4±2.8 bpm to 87.6±3.5 bpm after RMSG (p=0.1).

**Effect on spirometric values (Figs. 3, 4)**

The FVC was slightly increased after RMSG in 24 patients. The FVC of all 34 patients significantly increased from 1807±141 ml to 1923±145 ml (p<0.05). There was no significant change in FEV₁ (851±87 ml before; 888±88 ml after RMSG). On the maximal expiratory flow-volume curve, peak expiratory flow rate (PEFR) significantly increased from
Respiratory Muscle Stretching

Fig. 2. Immediate effect of respiratory muscle stretch gymnastics (RMSG) on dyspnea at rest. Before RMSG, 19 of 34 patients had dyspnea at rest on the 150-mm visual analog scale (VAS value > 0). In 14 of the 19 patients with dyspnea at rest VAS values were lower after RMSG. The VAS value of all 34 patients decreased from 11.6 ± 3.4 (mean ± SE) mm to 6.2 ± 2.2 mm (p < 0.01). Line of identity is shown.

Table 2. Mean blood pressure (BP), pulse rate (PR) and arterial blood oxygen saturation (SaO₂) before and after respiratory muscle stretch gymnastics (RMSG).

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<tr>
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<th>Systolic BP (mm Hg)</th>
<th>Diastolic BP (mm Hg)</th>
<th>PR (bpm)</th>
<th>SaO₂ (%)</th>
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<tr>
<td>Before RMSG</td>
<td>123.9 ± 3.5</td>
<td>80.1 ± 1.9</td>
<td>85.4 ± 2.8</td>
<td>95.7 ± 0.4</td>
</tr>
<tr>
<td>After RMSG</td>
<td>123.8 ± 3.3</td>
<td>78.7 ± 1.8</td>
<td>87.6 ± 3.5</td>
<td>95.8 ± 0.4</td>
</tr>
<tr>
<td>NS</td>
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Values expressed as mean ± SE; NS, not significant; bpm, beats per minute.

2.03 ± 0.27 L/sec to 2.26 ± 0.27 L/sec. Maximal flow at 50% of vital capacity (Vₑ₅₀) was 0.42 ± 0.07 L/sec before and 0.43 ± 0.07 L/sec after RMSG, and maximal flow at 25% of vital capacity (Vₑ₂₅) was 0.22 ± 0.03 L/sec before and 0.20 ± 0.03 L/sec after RMSG. There was no significant change in Vₑ₂₅ or Vₑ₅₀. There was no correlation between the decrease in dyspnea and improvement in the spirometry.

Discussion

The study results suggest that RMSG immediately reduces dyspnea at rest, and improves spirometric variables in patients with severe COPD.
Fig. 3. Immediate effect of RMSG on forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV1). FVC was significantly increased from 1807±141 ml to 1923±145 ml (p<0.01). FEV1 did not change significantly.

Fig. 4. Immediate effect of RMSG on maximal expiratory flow-volume curve. There was no significant (NS) difference in maximal flow at 50% of vital capacity (V50) and in maximal flow at 25% of vital capacity (V25) before and after RMSG. Peak expiratory flow rate (PEFR) was significantly increased from 2.03±0.27 L/sec to 2.26±0.27 L/sec (p<0.05).

On spirometry, FVC and PEFR but not FEV1 were significantly higher immediately after RMSG. It has been reported that 1) patients with COPD have lower chest wall compliance than normal subjects, 2) that vital capacity is correlated with compliance of the entire respiratory system, and 3) that vital capacity is reduced largely because of the diminished distensibility of the chest wall in patients with COPD. Although the chest wall compliance was not measured in the present study, it seems reasonable to assume that the patients with COPD in the present study had stiff chest walls, including the respiratory muscles. Hagbarth et al. reported that muscle stretching causes a decrease in finger flexor stiffness. Therefore, it is possible that respiratory muscle stretching similarly affected chest wall compliance and decreased chest wall stiffness. The increase in FVC and PEFR might also be explained by this mechanism. The lack of change in FEV1 may indicate that airway patency is not affected by RMSG.
In the present study, RMSG immediately decreased VAS ratings in patients with COPD. Because VAS measures dyspnea reproducibly both at rest and during exercise in patients with COPD, we believe that the decrease in VAS rating seen in the present study indicates a decrease in dyspnea at rest. Even though various physical conditioning and respiratory maneuvers, such as deep breathing and diaphragmatic breathing, have been shown to decrease dyspnea over time, to our knowledge no previous study has shown an immediate, measurable decrease in dyspnea. Thus, the immediate decrease in dyspnea observed in the present study is likely due to RMSG. This immediate dyspnea-decreasing effect of RMSG seems to be a unique feature of this respiratory program and may help encourage patients and keep compliance high. However, because the design of the present study was uncontrolled, the possibility of a placebo effect or nonspecific effects cannot be completely ruled-out. In the future, a controlled study should be carried out.

The mechanism whereby RMSG decreases dyspnea cannot be deduced from the present study, but increased afferent activity from contracting intercostal muscle spindles might be important in decreasing dyspnea. The IPV, or alternating vibration applied to the contracting intercostal muscle, decreases dyspnea. The effect has been suggested to be physiologic, since out-of-phase vibration, or alternating vibration applied to the noncontracting intercostal muscle, increases dyspnea. The dyspnea-decreasing effect of IPV is probably mediated by vibration-elicited afferent activity from chest wall respiratory muscle receptors to supraspinal centers. These afferents could be from muscle spindles, since vibration is a powerful stimulus of the muscle spindles. Stretching stimulates muscle spindles, and, due to alpha-gamma linkage, the sensitivity of muscle spindles is increased during contraction. Thus, stretching the contracting muscle is a powerful stimulus for the muscle spindles, and RMSG may increase afferent activity from the contracting chest wall respiratory muscle spindles in a manner similar to IPV. Kanamaru et al. recorded electromyographic activity from respiratory muscles and reported that activity during RMSG is greater than that during deep breathing alone, indicating that RMSG stimulates muscle spindles.

The BP, PR, and SaO₂ were measured for evaluation of clinical safety; there were no statistically significant changes in BP, SaO₂, or PR. These results suggest that RMSG itself causes little physical stress and can be a safe part of a pulmonary rehabilitation program, particularly in patients with severe impairments.

In summary, RMSG, designed to stretch the respiratory muscles predominantly during the contraction phase, decreased dyspnea at rest in patients with COPD. One session of RMSG may immediately decrease dyspnea at rest and improve pulmonary function. RMSG was performed safely and was effective in both elderly and severely impaired patients with COPD. By decreasing dyspnea, RMSG might help increase exercise capacity, activity of daily living, and quality of life in patients with COPD. We believe that RMSG is an effective and safe method of pulmonary rehabilitation.

Acknowledgment

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References


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