In-phase Chest Wall Vibration Decreases Dyspnea During Arm Elevation in Chronic Obstructive Pulmonary Disease Patients

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In-phase chest wall vibration (IPV) is known to decrease dyspnea in patients with chronic obstructive pulmonary disease (COPD) at rest and during leg exercise. In the present study, the effects of IPV (100 Hz) on dyspnea and arm fatigue during upper extremity activity were studied in 9 patients with COPD (mean FEV₁ 0.95 l). Dyspnea and arm fatigue (modified Borg scale) and ventilatory variables were measured during arm elevation (AE) with weights lifted straight above the head with and without IPV. Mean dyspnea during AE was 3.3 without IPV and 2.1 with IPV (p<0.05), but, arm fatigue, oxygen saturation and end-tidal FCO₂ were not affected by IPV. Minute ventilation during AE was significantly increased with IPV in 5 of 9 patients. The results suggest that IPV decreases dyspnea during AE.

Key words: obstructive, lung diseases, chest wall vibration, exercise, human, pulmonary emphysema, respiratory muscles

Introduction

Both the mechanism and management of dyspnea, in patients with chronic respiratory disease, remain unclear (1, 2). Regarding the management of dyspnea, it has been reported that chest wall vibration modifies the sensation of dyspnea. In-phase vibration (IPV, vibration of the contracting intercostal muscle, namely the inspiratory intercostal muscle during the inspiratory phase and the expiratory intercostal muscle during the expiratory phase) decreases dyspnea induced in normal subjects (3) and in chronic obstructive pulmonary disease (COPD) patients at rest and during leg exercise (4, 5). The alternate mode, 'out-of-phase vibration' (vibration of the non-contracting intercostal muscle, namely the inspiratory intercostal muscle during the expiratory phase and the expiratory intercostal muscle during the inspiratory phase) increases dyspnea (4, 6). The effect of IPV on dyspnea is hypothesized to involve stimulation of the muscle spindles, as IPV also increases tidal volume and decreases functional residual capacity, suggesting a tonic vibration reflex in the contracting intercostal muscles (4, 7, 8).

Method

Nine patients (8 males, 1 female) with moderate to severe COPD were studied (Table 1). They were naive to IPV, and their mean ± SD age and forced expiratory volume in one second (FEV₁) were 68 ± 6 year-old, and 0.95 ± 0.39 l,

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measurements at rest and at the end of AE with and without IPV (PULSOX-SP, Teijin, Tokyo). Dyspnea, fatigue and Spo2 were evaluated verbally as the resting value. Dyspnea and arm muscle fatigue were evaluated using a modified Borg (max 10) scale (12) each minute. AE was performed once without IPV and once with IPV. The trials were separated by at least a 15-minute rest and the sequence was random. The 1-minute measurement just preceding the first AE was considered as the resting value.

Dyspnea and arm muscle fatigue were evaluated verbally using a modified Borg (max 10) scale (12) each minute. AE was continued for 3 minutes and was stopped after the third evaluation or at the time when the patient wished to discontinue the trial. It was predetermined to terminate AE when the Borg scale reached 5, although this was not indicated to the patient prior to the study. Spo2 was recorded each minute by pulse oximetry (PULSOX-SP, Tejin, Tokyo). Dyspnea, fatigue and Spo2 measurements at rest and at the end of AE with and without IPV were compared and statistically analyzed with each other as a group. Standard ventilatory variables were measured breath-by-breath (AE280, Minato Ikagaku Co., Osaka) with the transducer attached to a face mask. These variables (tidal volume, minute ventilation, etc) of the 10 breaths recorded during the minute immediately preceding the first AE (resting values) and the 10 breaths recorded during the final minute of AE with and without IPV were compared and statistically analyzed with each other for each patient individually.

Results

Seven of the nine patients completed both 3-minute trials of AE. One patient requested termination of both trials after two minutes, and both trials were terminated after one minute in the other patient because the dyspnea scale reached 5.

Results from a typical patient are shown in Fig. 1. During the trial without IPV, dyspnea, arm fatigue and Ve increased with time. With IPV, the dyspnea rating was smaller, the fatigue rating remained unchanged, and Ve was larger than the trial without IPV. AE changed Spo2 from 97 to 96 without IPV and from 97 to 98 with IPV. Both are likely to be mere fluctuations and not significant changes. Changes in Fetco2 with AE and IPV also remained small.

Overall changes are shown in Figs. 2 and 3. The mean ± SD dyspnea at the end of AE was 3.3 ± 1.2 without IPV, and 2.1 ± 1.2 with IPV, which was significantly smaller (Wilcoxon paired test, p<0.05), but no significant difference was seen in arm fatigue (Fig. 2). Spo2 did not decrease during either trial of AE. Average Fetco2 at rest and at the end of AE without IPV and with IPV were 4.2, 4.4, and 4.3% respectively (Fig. 2). IPV did not have any systematic effect on either Spo2 nor Fetco2 during AE. Individually in 3 cases, Fetco2 during AE with IPV was significantly lower than Fetco2 during AE without IPV (Student’s t-test, p<0.05). The difference was, however, less than 0.2% in all 3 of the cases.

Ve at the end of AE was significantly (Student’s t-test, p<0.05) greater with IPV compared to that without IPV in 5 patients (Fig. 3). IPV significantly increased the tidal volume in one patient, and the respiratory rate in 3 patients. Overall, IPV did not affect VO2 nor VCO2 during AE. AE significantly increased VO2 (248.7 ± 40.6 ml/min at rest) to 324.0 ± 73.6 ml/min without IPV and to 325.4 ± 72.3 ml/min with IPV. VCO2 (208.5 ± 34.6 ml/min at rest) was also increased to 264.8 ± 70.2 ml/min without IPV and to 268.7 ± 66.1 ml/min with IPV.

The results regarding dyspnea (Fig. 2) indicate that all 5 patients whose Borg rating was larger than average during AE without IPV had a lower Borg rating with IPV (4.2 ± 0.4 without

Table 1. Patient Profile

<table>
<thead>
<tr>
<th>Patient code</th>
<th>Age/Sex</th>
<th>Height/BW</th>
<th>VC</th>
<th>FEV1.0</th>
<th>TLC</th>
<th>FRC</th>
<th>Pao2</th>
<th>Paco2</th>
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<tr>
<td>1</td>
<td>70/M</td>
<td>170/39</td>
<td>1.96</td>
<td>0.63</td>
<td>6.83</td>
<td>5.43</td>
<td>85.9</td>
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<tr>
<td>2</td>
<td>78/M</td>
<td>153/52</td>
<td>3.25</td>
<td>1.57</td>
<td>6.25</td>
<td>4.71</td>
<td>60.8</td>
<td>35.2</td>
</tr>
<tr>
<td>3</td>
<td>70/M</td>
<td>160/48</td>
<td>3.11</td>
<td>1.48</td>
<td>5.77</td>
<td>3.83</td>
<td>63.5</td>
<td>35.3</td>
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<tr>
<td>4</td>
<td>77/F</td>
<td>142/42</td>
<td>1.89</td>
<td>0.56</td>
<td>3.96</td>
<td>2.81</td>
<td>64.1</td>
<td>41.8</td>
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<tr>
<td>5</td>
<td>73/M</td>
<td>171/53</td>
<td>3.39</td>
<td>1.10</td>
<td>7.18</td>
<td>5.17</td>
<td>68.0</td>
<td>40.6</td>
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<td>6</td>
<td>62/M</td>
<td>158/53</td>
<td>3.84</td>
<td>0.78</td>
<td>6.37</td>
<td>4.00</td>
<td>72.9</td>
<td>38.8</td>
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<td>167/45</td>
<td>1.70</td>
<td>0.50</td>
<td>6.24</td>
<td>5.15</td>
<td>83.3</td>
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</tr>
<tr>
<td>8</td>
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<td>168/73</td>
<td>2.71</td>
<td>0.90</td>
<td>5.24</td>
<td>3.47</td>
<td>53.6</td>
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<tr>
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<td>164/57</td>
<td>2.90</td>
<td>0.88</td>
<td>5.70</td>
<td>3.70</td>
<td>77.9</td>
<td>49.2</td>
</tr>
</tbody>
</table>

VC: vital capacity, FEV1.0: forced expiratory volume in 1 second. TLC: total lung capacity, FRC: functional residual capacity.
In-phase Vibration and Arm Elevation

![Graphs showing time profile of dyspnea and arm fatigue, measured on a modified Borg scale (max 10), $\dot{V}_E$, $\text{SpO}_2$, and $\text{FetCO}_2$ in a typical patient (code 9 on Table 1) with and without in-phase vibration (IPV). Positive numbers indicate minutes after arm elevation. The 2-minute baseline values are indicated by the negative numbers.](image)

Figure 1. Time profile of dyspnea and arm fatigue, measured on a modified Borg scale (max 10), $\dot{V}_E$, $\text{SpO}_2$, and $\text{FetCO}_2$ in a typical patient (code 9 on Table 1) with and without in-phase vibration (IPV). Positive numbers indicate minutes after arm elevation. The 2-minute baseline values are indicated by the negative numbers.

IPV, $2.7 \pm 1.0$ with IPV, $p<0.05$). On the other hand, in the other 4 patients whose Borg rating was less than average during AE without IPV, the Borg rating remained unchanged with IPV ($2.3 \pm 1.0$ without IPV, $1.25 \pm 1.0$ with IPV). The clinical profile of the 5 patients with strong AE dyspnea was compared with the 4 patients with slight AE dyspnea. $\text{SpO}_2$ at rest, during AE without IPV and during AE with IPV was higher in the 5 patients with strong AE dyspnea ($96.2 \pm 0.8$, $95.4 \pm 0.5$, $96.6 \pm 0.9$, respectively) compared with the 4 patients with slight AE dyspnea ($92.8 \pm 2.5$, $93.0 \pm 2.7$, $92.3 \pm 2.4$, respectively). The other clinical profiles were similar between the two subgroups.

Discussion

The results show that IPV decreased dyspnea during lifting weights straight above the head, but did not affect arm fatigue, oxygen saturation and end-tidal $\text{FCO}_2$. $\dot{V}_E$ during the elevation was significantly increased with IPV in 5 of 9 patients. Previous studies have also indicated that IPV decreases dyspnea (3–5).

The decrease in dyspnea observed in the present study could be due to a decrease in one or more of the following: 1) sensitivity in the sensory system, 2) central respiratory motor command (13, 14), and 3) afferent signals from the respiratory-related receptors such as those in respiratory muscles, airway, lungs, and the chemoreceptors.

Sensory system

It is unlikely that IPV decreased dyspnea due to a reduction in the sensitivity in the sensory system. It has been shown that skin vibration increases pain threshold (15). However, if sensory system sensitivity reduction was the principal reason for the decrease in AE dyspnea, a similar effect could have occurred for AE muscle fatigue. This was not the case. Thus the reduction in AE dyspnea may be a more specific effect of IPV.

Central motor command

The intensity of dyspnea has been shown to be closely related to the level of ventilation and metabolism (16). Thus, the relationship between ventilation and dyspnea is often investigated when evaluating the effects of an intervention designed to relieve dyspnea. Reduction in $\dot{V}_E$ has been the proposed mechanism for decreased AE dyspnea after arm exercise training (10). $\dot{V}_E$ could be a reflection of the central respiratory motor command; however, this reflection of the central respiratory motor command by $\dot{V}_E$ may not be as good during IPV application compared to without IPV application. This is because IPV has been shown to elicit a spinal tonic vibration reflex, and increase muscle activation (7, 8). Thus, although IPV increased $\dot{V}_E$.
Figure 2. Dyspnea, arm fatigue, arterial oxygen saturation (Spo2) and end-tidal Fco2 (Fetco2) at the end of arm elevation (AE) with and without in-phase vibration (IPV). Asterisk indicates significant difference (p<0.05). Dyspnea rating at rest was 0.6 ±0.8, and arm fatigue rating was 0 in all patients. Neither AE nor IPV had a significant effect on either Spo2 or Fetco2.

Figure 3. Ventilatory variables at rest (R) and during AE with and without in-phase vibration (IPV).
during AE in 5 out of 9 patients, due to spinal tonic vibration reflex, the central respiratory motor command may have decreased, leading to a decrease in dyspnea.

**Afferent signals**

It is unlikely that IPV decreased AE dyspnea chiefly due to a reduction in the chemical stimulation. Hypoxemia and hypercapnia are thought to be related to dyspnea (2). However, in the present study, the deterioration in blood gas with AE was very slight, if any and it was unlikely to have been the cause of AE dyspnea. Also, there was no overall improvement in Spo2 during AE with IPV. A decrease in Fetco2 with IPV was observed in 3 patients, however as the decrease was less than 0.2% in all 3 patients, it was unlikely to have decreased AE dyspnea. Thus, improvement in blood gas is unlikely to be the principle reason for the decrease in AE dyspnea with IPV. The comparison between the 3 patients with strong AE dyspnea and 4 patients with slight AE dyspnea supports this conclusion. Interestingly, Spo2 at rest and during AE with or without IPV in the patients with strong dyspnea was higher than in the patients with slight dyspnea. This indicates that desaturation was not the chief factor that determined AE dyspnea and also that improvement in desaturation can not explain the effect of IPV.

The muscle spindles in the upper tonically active inspiratory muscles (9), may be powerfully stimulated when extended during the expiratory phase. Such afferents would be similar to those elicited by 'out-of-phase' vibration (4, 6), and are considered to be a possible cause of AE dyspnea (9). It is possible that IPV decreased such 'out-of-phase' afferents, as vibration applied to the expiratory intercostal muscle has been shown to inhibit inspiratory activity (7). Also, increased firing from the contracting intercostal muscles might have decreased dyspnea as well (4).

Vagal afferent signals from pulmonary mechanoreceptors have also been suggested to be involved in dyspnea (17, 18). IPV could have stimulated the pulmonary mechanoreceptors either directly or due to the enhanced ventilation. However, the role of vagal afferent signals in dyspnea, if any, is probably enhancement (17, 18). Thus, it is unlikely that IPV decreased dyspnea by decreasing vagal afferent signals.

In COPD patients, upper inspiratory intercostal muscles are suggested to be tonically active during AE (9), and may contribute to the increase in functional residual capacity (FRC) (19). Increasing hyperinflation during leg exercise has been indicated to be related to dyspnea in COPD patients (20). Since IPV may stimulate the muscle spindles of the expiratory intercostal muscle during the expiratory phase, elicit tonic vibration reflex (7) and decrease FRC at rest in COPD patients (4), a decrease in FRC could have also occurred with IPV and contributed to the decrease in dyspnea in the present study.

In conclusion, O2 desaturation may not be involved in AE dyspnea. The present study does not support O2 supplementation against dyspnea during arm elevation in patients with COPD. On the other hand, IPV specifically decreases dyspnea during AE without decreasing ventilation or improving blood gas. This effect may in part involve afferent signals from intercostal muscle spindles.

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**References**