Low frequency breathing at rest and during exercise in severe chronic obstructive bronchitis

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ABSTRACT The effect of low frequency breathing compared with spontaneous breathing was examined at rest and during exercise (40 watts) in 12 patients suffering from severe chronic obstructive bronchitis. At rest low frequency breathing improved significantly the alveolar ventilation and the tensions of oxygen and carbon dioxide in the arterial blood. There was no significant change in ventilation minute volume. During exercise low frequency breathing significantly decreased ventilation minute volume, and there was no significant improvement in gas exchange. The decrease in ventilation during low frequency breathing at 40 watts compared with spontaneous breathing at the same lung volume was due to expiratory flow limitation. The findings suggest that this technique may impair exercise tolerance in patients with severe chronic obstructive bronchitis.

Low frequency breathing (LFB) has been recommended for patients suffering from chronic obstructive bronchitis. At rest this pattern of breathing, characterised by low respiratory frequency and increased tidal volume improves alveolar ventilation and therefore gas exchange (Motley, 1963; Lockhart, et al, 1966; Paul et al, 1966; Gimenez, 1968; Sergysels et al, 1973). The effect, however, lasts only for the time the new ventilation pattern is maintained, and it is unclear how many patients will be able to adopt the pattern in their daily life. In particular patients suffering from airways obstruction whose ventilation approaches their maximal ventilatory capacity during moderate exercise (Cotes, 1965) may have difficulty preserving their ventilation during breathing with reduced frequency. To examine this aspect 12 patients suffering from severe chronic obstructive lung disease were trained to lower their respiratory frequency at rest and during exercise on a cycle ergometer. Ventilation (V̇), alveolar ventilation (VA), and gas exchange were measured during spontaneous and during low frequency breathing.

Methods

The 12 patients included in the study were all suffering from chronic obstructive bronchitis; most of them showed radiological evidence of emphysema. The ventilatory capacity was impaired. At rest, the patients were hypoxic (Pao2 < 70 mmHg), and nine of them were hypercapnic (Paco2 > 45 mmHg). These and other details of the subjects are given in table 1. Values for lung volumes were related to the reference values of Grimby and Söderholm, 1963.

After local anaesthesia, a cannula was inserted in the brachial artery for measuring Pao2, Paco2, and pH (Radiometer). Ventilation (V̇), respiratory frequency (fR), and tidal volume (Vt) were measured using a Fleish pneumotachograph. The mixed expired gas was analysed for CO2 and O2 using respectively Jaeger and Servomex gas analysers. From these data the O2 uptake and CO2 output (respectively V̇O2 and V̇CO2) and the relation of the alveolar to the total ventilation (VA/V̇) were calculated. At rest during spontaneous breathing, the measurements were made under steady state conditions at the end of an eight-minute period of observation. The patients then lowered their respiratory frequency (to about 8 min⁻¹), and the measurements were repeated.

The subjects were subsequently exercised on a cycle ergometer (Jaquet) at a load of 40 watts with first spontaneous breathing over eight minutes and then, after a 20-minute rest, with LFB. Tidal volume was again roughly doubled but respiratory frequency was never lower than 10 min⁻¹.

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Table 1 Spirometric values and blood gases at rest. Values for lung volumes are referred to normals established by Grimby (mean and SD; n=12)

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>VC (% predicted)</th>
<th>TLC (% predicted)</th>
<th>RV (% predicted)(ml)</th>
<th>FEV₁ (% predicted)</th>
<th>FEV₁/VC</th>
<th>Raw cm H₂O l/s</th>
<th>pH</th>
<th>PaCO₂ (mmHg)</th>
<th>PaO₂ (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>52-6</td>
<td>63-4</td>
<td>97-5</td>
<td>184-3</td>
<td>1025</td>
<td>35-5</td>
<td>6-25</td>
<td>7-38</td>
<td>47-6</td>
</tr>
<tr>
<td>SD</td>
<td>8-7</td>
<td>15-9</td>
<td>18-0</td>
<td>32-1</td>
<td>360</td>
<td>8-2</td>
<td>1-69</td>
<td>0-03</td>
<td>5-3</td>
</tr>
</tbody>
</table>

Results

Table 2 summarises the results obtained at rest and during exercise during spontaneous and low frequency breathing.

At rest
With LFB the mean respiratory frequency was lowered from 21.9 min⁻¹ to 10.2 min⁻¹ and the tidal volume increased from 544.6 ml to 1122 ml, but total ventilation remaining unchanged.

Tidal volume expressed as a percentage of FEV₁ and of VC was respectively 58.3% and 20.3% during SB and 110.2% and 38.1% during LFB.

VO₂ remained unchanged while VCO₂ increased during LFB.

With LFB, VA and VA increased significantly, leading to a significant increase in PaO₂ and decrease in PaCO₂.

Exercise
Exercise induced an increase in VA and VA but despite this during SB most patients incurred a small decrease in PaO₂ and increase in PaCO₂. During LFB, Vt was again roughly doubled going from 877 ml to 1552 ml while frequency was lowered from 28.7 min⁻¹ to 15.3 min⁻¹. Ventilation minute volume decreased from 24.5 l/min to 21.6 l/min Vt % and Vt % were respectively 90.3% and 31.4% during SB and reached 153.1% and 53.7% during LFB.

Changes in VO₂ and VCO₂ were not significant. LFB resulted in a small increase in VA and slight reduction in PaCO₂. There was no significant change in PaO₂.

Discussion

This study confirms the finding of others that at rest LFB with or without pursed lips increases the alveolar ventilation and this improves gas exchange of respiratory patients with chronic hypoxia and hypercapnia (Lockhart et al, 1966; Thoman et al, 1966; Sergysels et al, 1973). A similar improvement has been reported during exercise (Jimenez, 1968), but the patients had less impairment of lung function than in this study.

During pursed lip breathing Mueller et al (1970) obtained similar findings at rest. During exercise ventilation fell from 27.6 l min⁻¹ to 22.8 l min⁻¹, and there was no significant change in PaO₂ or PaCO₂.

During LFB, patients in our study were able to bicycle for eight minutes at 40 watts without subjective discomfort. Only two of the 12 patients, however, were able to maintain the ventilation at its normal level. Five of the 12 patients showed a significant

Table 2 Data obtained at rest and during exercise (40 watts) with spontaneous breathing (SB) and low frequency breathing (LFB). Mean and SD are presented; values at rest and at 40 watts are statistically analysed with paired t test

<table>
<thead>
<tr>
<th></th>
<th>V̇E (l/min)</th>
<th>f</th>
<th>V̇L (ml/min)</th>
<th>V̇O₂ (ml/min)</th>
<th>V̇CO₂ (ml/min)</th>
<th>VA (l)</th>
<th>VA/VA (%)</th>
<th>pH</th>
<th>PaCO₂ (mmHg)</th>
<th>PaO₂ (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SB</td>
<td>Mean 24-500</td>
<td>28-7</td>
<td>877-0</td>
<td>698</td>
<td>662</td>
<td>11-326</td>
<td>47-1</td>
<td>7-34</td>
<td>51-6</td>
<td>56-0</td>
</tr>
<tr>
<td>SD</td>
<td>3-400</td>
<td>6-6</td>
<td>220-0</td>
<td>79</td>
<td>114</td>
<td>2-824</td>
<td>10-4</td>
<td>0-02</td>
<td>6-7</td>
<td>10-2</td>
</tr>
<tr>
<td>At rest LFB</td>
<td>Mean 21-580</td>
<td>15-3</td>
<td>1552-0</td>
<td>722</td>
<td>721</td>
<td>12-868</td>
<td>58-7</td>
<td>7-34</td>
<td>49-7</td>
<td>56-0</td>
</tr>
<tr>
<td>SD</td>
<td>3-220</td>
<td>5-3</td>
<td>497-0</td>
<td>103</td>
<td>165</td>
<td>4-070</td>
<td>13-4</td>
<td>0-04</td>
<td>6-3</td>
<td>9-6</td>
</tr>
<tr>
<td>p</td>
<td>&lt; 0-0005</td>
<td>&lt; 0-0005</td>
<td>&lt; 0-005</td>
<td>NS</td>
<td>NS</td>
<td>&lt; 0-0125</td>
<td>&lt; 0-0005</td>
<td>NS</td>
<td>&lt; 0-025</td>
<td>NS</td>
</tr>
</tbody>
</table>
increase in VA while in two there was a decrease. On average LFB increased VA by 36% at rest but only by 20% during exercise. The absence of a worthwhile improvement in gas exchange could be explained at least in part by the relative decrease in ventilation.

Grimby and Stiksa (1970) showed that patients with chronic obstructive bronchitis increased their end expiratory level during exercise and on this account were able to generate higher expiratory flows. This tendency would be modified by low frequency breathing when, as in this study, Vt reached 53% of VC. In one subject flow—volume loops recorded during spontaneous breathing at rest and during exercise with spontaneous and low frequency breathing were located in a maximal flow volume curve obtained during forced vital capacity (FVC) manoeuvre (see figure). Exercise with spontaneous breathing induced an upwards shift of the end expiratory level. LFB lowered the end expiratory level nearly to the FRC level observed at rest during spontaneous breathing.

At isovolume and therefore at isodriving force, flows recorded during spontaneous breathing both at rest and on exercise are higher than during forced expiration. During exercise, expiratory flows were higher with spontaneous than with low frequency breathing. At the same time the ventilation decreased from 29·5 l min⁻¹ to 20·4 l min⁻¹.

Ventilation can be expressed as the product of VT × TE × 60 where TE is the expiratory time and TE/Ttot the sum of TE and the inspiratory time (TI). VT and TE were respectively 0·77, 0·64 during VT/Ttot spontaneous and 0·44, 0·74 during low frequency breathing (see figure). Clearly, the decrease in VT/TE (that is the mean expiratory flow) was the determining factor of the relative limitation of V observed with LFB.

These findings show that lowering the respiratory frequency during exercise in patients with severe chronic obstructive bronchitis may not result in the expected improvement in gas exchange; instead there may be relative hypoventilation for mechanical reasons. This is due to the associated reduction in resting respiratory level diminishing the airways calibre and the force available for expiration. Thus any patient suffering from chronic airways obstruction should be investigated from this point of view before being instructed to breathe slowly during daily activities such as walking or climbing stairs.

References


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