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The Effect of Cold Air Inhalation on Respiratory Gas Exchange During Exercise in Patients With Chronic Obstructive Pulmonary Disease*

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This study was done to determine whether gas exchange is affected, both at rest and with exercise, by airflow resistance alterations previously observed during cold air breathing in patients with chronic obstructive lung disease with a history of sensitivity to cold air. Nine patients with cold air sensitivity were studied at rest and during treadmill exercise breathing room temperature and −12°C air. Seven of the nine patients had more distress during cold air breathing, but there were no significant differences in heart rates, O₂ consumption and arterial blood P₀₂ and P₇CO₂ between room temperature and −12°C air breathing, either at rest or during exercise. These results give support to the concept that the most marked change in air flow resistance during cold air breathing occurs in poorly ventilated lung areas.

INTRODUCTION

A change in environmental temperature may initiate the onset of respiratory symptoms such as cough, chest discomfort or dyspnea in patients with bronchial asthma, chronic bronchitis and pulmonary emphysema who are known to have a history of sensitivity to cold. These symptoms may be exaggerated by increasing activity in the cold.

Periodic measurements of pulmonary function in patients with chronic obstructive pulmonary disease have suggested that airway resistance increases as temperature decreases.1−4 Simonsson and co-workers5 found that air cooled to −20°C resulted in an increase in airway resistance in some patients with asthma or bronchitis. Wells and co-workers6 found that only those patients with a history of cold sensitivity have an increase in airway resistance during cold air inhalation. Studies previously reported from this laboratory7 indicate that the most marked change in airflow resistance during cold air breathing occurs in poorly ventilated lung areas and may result in airway closure in such lung areas. The previous studies did not indicate, however, how these airflow resistance changes during cold air breathing affect gas exchange in the lung.

This study was done to determine whether arterial blood gas tensions are affected by the airflow resistance alterations which were observed previously during cold air breathing, not only at rest, but also during exercise, when the symptoms are usually much more exaggerated. Nine patients with chronic obstructive pulmonary disease, and a known history of sensitivity to cold air, were studied at rest and during treadmill exercise, breathing air at room temperature and at −12°C.

METHODS

All studies were done in an air-conditioned laboratory with a mean temperature of 25°C±2°C. The breathing apparatus was arranged as shown in Figure 1. Compressed gas from a cylinder was allowed to flow through a coil of copper tubing (L₁). Immersion of this coil in the liquid nitrogen contained in the refrigerator allowed cooling of the inspired air to −12°C. The air source was connected to a Collins five-way valve (R). A two-way Douglas (D) valve with mouthpiece (M) was attached to the central portion of the Collins valve. A Douglas bag (B) was connected to the expiratory side of the valve through a two-way stopcock (ST) and was used to collect expired air for the determination of minute volume and oxygen and carbon dioxide concentrations. A J-valve (J) was used between the Collins valve and the source of inspired air so that the system could be operated with an excess flow which was vented to the outside through this J-valve. Check of the apparatus showed that none of the excess flow passed on through the system into the Douglas bag, which, had it...
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A Courand needle was placed in the brachial artery under local procaine anesthesia. Electrocardiographic leads were placed as described by Abarquez et al.\textsuperscript{8} to record heart rate and to detect the occurrence of premature ventricular contractions. All measurements were made with the subject standing or walking at 1.5 mph on a flat treadmill. Inspired air was obtained from the compressed air system as described in Figure 1. Respiratory rate was counted by noting the movements of the patient's chest wall during the sampling period. The patients were studied initially breathing room temperature compressed air. They were allowed to breathe through the mouthpiece assembly for a five minute period prior to collection of the standing resting sample. Expired gas was collected in a Douglas bag and an integrated arterial blood sample (collected slowly and evenly in a 20 ml syringe over the entire five minute period) was withdrawn for Pco\textsubscript{2} and pH analyses. The ECG was recorded and respiratory rate determined. At the end of this collection period, the patient, breathing continuously through the mouthpiece, began to walk at 1.5 mph. The patient was allowed to rest for 15 minutes and the measurements were repeated at rest and during exercise with the patient breathing \(-12^\circ\text{C}\) air continuously.

Expired gas was analyzed for oxygen and carbon dioxide tension and arterial blood for Pco\textsubscript{2}, pH and Po\textsubscript{2} by Instrumentation Laboratories' gas analyzer using standard techniques. Percentage oxygen saturation was determined by the spectrophotometric method of Hickam and Frayser.\textsuperscript{9} The electrocardiogram was analyzed for heart rate and any abnormalities.

All patients had routine pulmonary function studies done on the day of the test. Functional residual capacity and airway resistance were measured using the whole body plethysmograph.\textsuperscript{10,11} Maximal voluntary ventilation was determined using a low resistance two-way valve.

RESULTS

Pulmonary function data for nine patients (age range, 50-72 years) are summarized in Table 1. All patients showed obstructive ventilatory insufficiency as evidenced by a decrease in vital capacity and maximal voluntary ventilation, and an increase in airway resistance. Functional residual capacity, Table I—Pulmonary Function Data in 9 Patients With Chronic Obstructive Pulmonary Disease

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yrs)</th>
<th>Body Surface Area (M\textsuperscript{2})</th>
<th>Vital Capacity (liters)</th>
<th>Functional Residual Capacity (liters)</th>
<th>Residual Volume (liters)</th>
<th>Total Lung Capacity (liters)</th>
<th>RV/TLC (X 100)</th>
<th>Maximum Voluntary Ventilation (liters/min)</th>
<th>Airway Resistance (cmH\textsubscript{2}O/L/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50</td>
<td>1.76</td>
<td>1.78</td>
<td>5.40</td>
<td>4.52</td>
<td>6.30</td>
<td>71.7</td>
<td>19</td>
<td>18</td>
</tr>
<tr>
<td>2</td>
<td>58</td>
<td>2.11</td>
<td>3.27</td>
<td>4.50</td>
<td>4.03</td>
<td>7.30</td>
<td>55.2</td>
<td>71</td>
<td>60</td>
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<tr>
<td>3</td>
<td>63</td>
<td>2.02</td>
<td>1.50</td>
<td>3.90</td>
<td>3.79</td>
<td>5.29</td>
<td>71.6</td>
<td>40</td>
<td>37</td>
</tr>
<tr>
<td>4</td>
<td>70</td>
<td>1.92</td>
<td>2.40</td>
<td>4.00</td>
<td>3.47</td>
<td>5.87</td>
<td>59.1</td>
<td>44</td>
<td>46</td>
</tr>
<tr>
<td>5</td>
<td>58</td>
<td>2.10</td>
<td>2.06</td>
<td>2.80</td>
<td>7.07</td>
<td>9.73</td>
<td>78.8</td>
<td>23</td>
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<tr>
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<td>78.7</td>
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<tr>
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<td>1.98</td>
<td>3.39</td>
<td>5.50</td>
<td>4.43</td>
<td>7.82</td>
<td>56.6</td>
<td>48</td>
<td>48</td>
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<tr>
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<td>3.78</td>
<td>4.30</td>
<td>2.30</td>
<td>6.06</td>
<td>37.8</td>
<td>58</td>
<td>59</td>
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<tr>
<td>9</td>
<td>72</td>
<td>1.51</td>
<td>2.71</td>
<td>4.40</td>
<td>3.41</td>
<td>6.12</td>
<td>55.7</td>
<td>39</td>
<td>53</td>
</tr>
</tbody>
</table>

Mean: 60.9    1.91    2.49    5.12    4.27    6.80    62.8    40.3    39.7    5.6

±S.D. 8.1    .65    .94    1.35    1.66    2.60    13.5    17.5    17.7    2.5

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residual volume and the ratio RV/TLC were increased.

The results for the cardiopulmonary parameters at rest and during exercise breathing air at room temperature and at \(-12^\circ C\) are shown in Tables 2 and 3. Heart rate, respiratory rate, minute ventilation and oxygen consumption were not significantly changed by inhalation of cold air at rest, but all were significantly \((p < .01)\) higher during exercise (Table 2). Minute ventilation was significantly \((p < .05)\) reduced by cold air breathing during exercise, but heart rate, respiratory rate and oxygen consumption were not different from exercise while breathing air at room temperature.

At rest, breathing air at \(25^\circ C\), mean arterial blood values were: \(P_{CO_2}, 45 \pm 7\) mmHg, \(P_{O_2}, 70 \pm 10\) mmHg and \(pH, 7.40 \pm .03\) (Table 3). There was no statistically significant change in these values with breathing air at \(-12^\circ C\). Compared to rest, exercise resulted in a significant \((p < .05)\) reduction in arterial \(P_{O_2}\) (63.6 mmHg) and in percentage \(O_2\) saturation, both breathing room temperature and \(-12^\circ C\) air, but no statistically significant change in \(P_{CO_2}\). There were no significant changes in either \(P_{CO_2}\), \(P_{O_2}\) or \(pH\) from exercise breathing room temperature air to exercise breathing \(-12^\circ C\) air.

Two of the nine patients (patients 2 and 7) developed premature ventricular contractions on breathing cold air at rest as well as during exercise. Two others (4 and 8) had this abnormality throughout the study. Seven of the nine patients studied complained of dyspnea and cough following cold air inhalation at rest and exercise; four developed wheezing. Although there was no significant difference in the duration of exercise between warm and cold air, all subjects indicated that they felt it more difficult to walk while breathing \(-12^\circ C\) air.

**Discussion**

Previous studies in this laboratory\(^7\) and those of
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others\(^6\) indicated that normal subjects and patients who give no history of symptoms in cold are not affected by breathing cold air. Therefore, only patients with a history of symptoms of increased respiratory difficulty in cold air were chosen for this study. The primary changes during cold air breathing appear to be in air-flow resistance\(^5,\)\(^6\) and our previous work\(^7\) indicates that the most marked change in air-flow resistance during cold air breathing occurs in poorly ventilated lung areas, possibly resulting in complete airway closure in some areas. The present study was done to determine how gas exchange and arterial blood gases are affected by these air-flow resistance changes which have been shown to occur during cold air breathing.

Air at \(-12^\circ\text{C}\) was used in this study rather than the \(-40^\circ\text{C}\) used in the previous study\(^7\) because it is a temperature to which patients in the local climate are more frequently exposed. The amount of exercise which could be tolerated by these patients was enough to double the oxygen consumption and produce a significant reduction in arterial oxygen saturation.

In spite of increased respiratory distress and previously shown air-flow resistance changes during cold air breathing, arterial blood \(\text{Po}_2\), \(\text{PCO}_2\) and pH were not changed by cold air breathing, either at rest or during exercise. The development of wheezing and dyspnea during cold air inhalation support the suggestion that bronchoconstriction may have occurred. The absence of a significant change in blood gas values, either at rest or with exercise while breathing cold air, may reflect a preferential shifting of alveolar ventilation from poorly ventilated areas.

The increased subjective sensation of respiratory distress on breathing cold air may be mediated through a reflex mechanism, which is responsive to the increased work of breathing or which occurs because of stimulation of unknown receptors within the tracheo-pharynx. Reflex inhibition of respiration and of spontaneous muscular action potentials from accessory respiratory muscles has been seen in animals in response to vaporized stimuli.\(^12,\)\(^13\) Simonsson and co-workers\(^8\) showed that the airway resistance increase which occurred in some patients with asthma or bronchitis during inspiration of \(-20^\circ\text{C}\) air could be prevented by administration of atropine sulfate, which blocked efferent cholinergic pathways. They suggest that sensitized cough receptors may be involved in triggering reflex airway constriction in such patients. A reflex mechanism has been postulated by Sorensen\(^14\) to explain the high incidence of angina pectoris following cold exposure.

This study emphasizes the fact that in spite of the increased severity of respiratory symptoms associated with air-flow resistance changes during cold air breathing in some patients with chronic obstructive lung disease, arterial blood \(\text{PO}_2\), \(\text{PCO}_2\) and pH are not affected.

References


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