Rate of Decay or Increment of PaO$_2$
Following a Change in Supplemental Oxygen in Mechanically Ventilated Patients With Diffuse Pneumonia*

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It has been shown that patients with COPD require as long as 20 min for equilibration of oxygen tension to occur after changing the fraction of inspired oxygen (FIO$_2$). To date, there have been no studies to determine the equilibration time for the PaO$_2$ in mechanically ventilated patients with diffuse pneumonia. We studied seven patients (five males, two females) with radiographic evidence of diffuse pneumonia. All patients required mechanical ventilation. After introducing a change in FIO$_2$, arterial blood gas values were measured at 5-min intervals for 30 min. Four patients achieved maximal change in PaO$_2$ after 5 min, while four patients required 10 min. These results are similar to those found in patients with left ventricular failure who experience equilibration rapidly; however, patients with COPD experience it at a much slower pace. These observations have clinical importance when managing unstable patients where time is a critical element. (Chest 1993; 103:554-56)

Mechanical ventilation is used frequently as a life-supportive procedure in patients with respiratory failure. Profound hypoxia often is a major feature of acute lung disease and therapy often involves oxygen supplementation with mechanical ventilation. In order to monitor the mechanically ventilated patient, physicians are called upon to interpret arterial blood gas values. How does one know when to repeat an arterial blood gas determination once a change in the fraction of inspired oxygen (FIO$_2$) is made? It has been suggested that a physician wait 15 to 30 min after changing or discontinuing supplemental oxygen before sampling the PaO$_2$. Comparative studies were performed by two groups. The first was done by Howe et al.; they studied serial blood gas determinations in patients with varying types of heart disease before and after discontinuation of supplemental oxygen. All patients were free of pulmonary disease. The PaO$_2$ values returned to baseline within 7 min after supplemental oxygen was withdrawn. The second was conducted by Sherter et al., who studied serial blood gas values in patients with moderate to severe COPD after discontinuation of oxygen. Their results demonstrated that patients who inhaled 100 percent oxygen, for a short period of time, required approximately 20 min for the PaO$_2$ level to return to baseline.

It is generally recommended that the first arterial blood gas sample should be drawn approximately 30 min after initiation of ventilator support regardless of the nature of the illness causing respiratory failure. However, since time is of the essence in critically ill patients, a 30-min wait may be unnecessary if equilibration were to occur more rapidly, such as in patients with left ventricular failure. Similarly, different lung disorders may have different rates of equilibration.

To date, there have been no studies to determine the time required for the PaO$_2$ to equilibrate in mechanically ventilated patients with diffuse pneumonia following a change in FIO$_2$. The present study was designed to assess the time course of the descent or ascent of PaO$_2$ after a change in FIO$_2$ is made in mechanically ventilated patients with diffuse pneumonia.

MATERIALS AND METHODS

Clinical Data

Seven patients with clinical and radiographic evidence of diffuse pneumonia were studied. There were five male and two female patients ranging in age from 27 to 85 years. None of the patients had any significant underlying obstructive lung disease.

All patients had indwelling arterial cannulae and were maintained on MA-2 ventilators. Arterial blood gas analyses were performed using an Instrument Laboratory 1312 blood gas analyzer. An arterial blood gas sample was drawn into a heparinized plastic syringe. Samples were immediately placed in ice and within 1 min blood gas analyses were performed by one of the investigators.

Changes in FIO$_2$ were ordered by individual physicians managing these seven patients. We measured arterial blood gas values prior to the change in FIO$_2$ and thereafter at 5-min intervals for a 30-min period. No other instrument parameters were changed (i.e., rate, tidal volume, positive end expiratory pressure or mode of mechanical ventilation) during the course of each individual experiment. Alveolar-arterial oxygen gradient was calculated for each patient for each arterial blood gas sample obtained. The study was approved.

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Table 1—Clinical Characteristics for Seven Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, yr</th>
<th>Sex</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27</td>
<td>F</td>
<td>HIV+, bilateral pneumonia, culture-negative</td>
</tr>
<tr>
<td>2</td>
<td>65</td>
<td>M</td>
<td>Chronic lymphocytic leukemia, bilateral pneumonia, culture-negative</td>
</tr>
<tr>
<td>3</td>
<td>35</td>
<td>M</td>
<td>HIV+, bilateral pneumonia, culture-negative</td>
</tr>
<tr>
<td>4</td>
<td>51</td>
<td>M</td>
<td>Bilateral pneumococcal pneumonia via transbronchial biopsy</td>
</tr>
<tr>
<td>5</td>
<td>85</td>
<td>M</td>
<td>Bilateral <em>Pseudomonas aeruginosa</em> pneumonia, pulmonary edema; Swan-Ganz wedge-20</td>
</tr>
<tr>
<td>6</td>
<td>34</td>
<td>M</td>
<td>HIV+, bilateral pneumonia, culture-negative</td>
</tr>
<tr>
<td>7</td>
<td>32</td>
<td>F</td>
<td>HIV+, bilateral <em>Staphylococcus aureus</em> pneumonia</td>
</tr>
</tbody>
</table>

by the Human Use Committee of our Institution.

Statistical Analysis

To facilitate comparison of PaO₂ for any sample drawn after the therapeutic intervention with its appropriate control (namely, PaO₂ for the same subject prior to adjustment of FIO₂), we arbitrarily assigned the value of zero to the control sample and the number 100 to the 30-min sample. The number 100 was arbitrarily assigned to the 25- and 30-min samples of the subject who had only five samples drawn. All other samples were assigned a number between 0 and 100 which represented a ratio of change for that sample (relative to control) divided by 100. Two-way analysis of variance was performed; significance of difference was then tested by the Newman-Kuels test.

RESULTS

Clinical characteristics for the seven patients are presented in Table 1. There were five men and two women studied. The average age of the subjects tested was 47 years (range, 27 to 85 years). All patients had diffuse, bilateral pneumonia infiltrates on a chest x-ray film. Four patients had a history of human immunodeficiency virus (HIV) infection. One patient had a history of chronic lymphocytic leukemia. Three of the seven patients had culture-proven bacterial infection (pneumococcal pneumonia, *Pseudomonas aeruginosa* and *Staphylococcus aureus*). All patients required 30 to 40 cm H₂O peak airway pressure to deliver the prescribed tidal volume.

Individual arterial gas tensions are presented in Table 2. There are eight sets of data provided because one subject was studied twice. Data expressed as percentage of total change appear in Figure 1. Five minutes after intervention, the mean change was 87±5 percent. The 10-min samples were, on the average, 103±7 percent. Analysis of variance showed that the difference for the mean values of percentage of change between sample times was significant at the 0.001 level. Comparison testing showed that the mean of control sample was different from all other samples (p<0.05). There were, however, no differences between the means of percent change at any other two sample times after the change of FIO₂ was instituted.

Analysis of variance showed that the difference for percentage of change between subjects was significant at the 0.01 level. There were no significant differences in the arterial Pco₂ between the initial value and those obtained throughout the experiment in each case, thus eliminating the possibility of a ventilatory change influencing the PaO₂.

Table 2—Serial Measurements of Blood Oxygen Tension in Eight Study Trials

<table>
<thead>
<tr>
<th>Patients</th>
<th>Before Therapeutic Intervention</th>
<th>After Therapeutic Intervention</th>
<th>Minutes After Changes in FIO₂, mm Hg</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>1a</td>
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<td>131</td>
<td>92</td>
</tr>
<tr>
<td>1b</td>
<td>60</td>
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<td>71</td>
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</table>
DISCUSSION

Results from these studies in seven patients showed that in spite of obvious severe diffuse intrapulmonary disease, small changes of FIO₂ were rapidly followed by parallel changes in PaO₂. This rapid response, also seen by other investigators in normal subjects and in patients with mild heart failure, is different from the response observed in patients with chronic or acute airway obstruction. In this latter group, as much as 20 min may be required for stabilization of PaO₂ after a change in FIO₂. Recognition of the difference in response rate between patients with COPD and those with diffuse pneumonia is important for patient management as it permits sampling of arterial blood gas values with greater precision, thus eliminating guesswork and waste of valuable time.

We believe it unlikely that our patient population would fulfill pathologic criteria for the adult respiratory distress syndrome because in this latter group changes in FIO₂ are accompanied by little, if any, change in PaO₂. This is explained by the fact that hypoxemia in adult respiratory distress syndrome is caused by intrapulmonary shunt, whereas hypoxemia in most other conditions of cardiopulmonary disease is the result of mismatching of the distributions of ventilation with perfusion (V/Q) in the lung.

This difference in the quality of gas exchange between patients with diffuse nonobstructive disease and patients with airway obstruction is easily explained by consideration of V/Q relationships previously described in patients with a variety of lung disorders. In the lungs of patients with interstitial lung disease, very little blood flows to regions of low V/Q or shunt. Furthermore, regions of high V/Q do not exist. Since most inspired air and most venous blood goes to the lung regions with normal V/Q, the process of gas exchange often is efficient in patients with interstitial lung disease or diffuse pneumonia in spite of profound anatomic and mechanical abnormalities of the lung. In contrast, regions of high and low V/Q commonly are found in patients with airway obstruction and may account for the inefficiency of gas exchange seen in these patients. This difference becomes crucial when managing unstable mechanically ventilated patients where time is of the essence.

REFERENCES