The First Few Hours Off a Respirator*

Robert Gilbert, M.D.;** J. H. Auchincloss, Jr., M.D.;**
David Peppi, B.S.;† and Kumar Ashutosh, M.D.‡

Fourteen patients receiving prolonged assisted ventilation were studied during their first trial period off the respirator. To avoid artifacts due to valves, mouthpieces, and noseclips, tidal volume off the respirator was measured continuously by electromagnetic sensors which detected changes in the anterior and posterior diameter of the rib cage and abdomen. Within five minutes of withdrawal from the respirator tidal volume fell, often to surprisingly low values, and respiratory frequency rose. Mean ventilation did not change. The changes in tidal volume, respiratory frequency, and heart rate did not indicate the changes which were occurring in the arterial blood gas levels. Furthermore, the magnitude of the changes in the breathing pattern did not predict whether or not the trial off the respirator would be successful. The changes in tidal volume, respiratory frequency, heart rate and the blood gas values did not correlate well with the subjective sensation of dyspnea.

The first attempt to remove a patient from a respirator after a period of assisted ventilation is a clinical experiment. The patient is carefully observed, subjective responses noted, and the vital signs monitored. Arterial blood gas analysis is usually considered a crucial part of the evaluation.¹ To our knowledge, however, no data are available concerning the expected time course of the vital signs and blood gas levels in the immediate period away from the respirator. Furthermore, the relationships between changes in respiratory frequency (f), tidal volume (VT), minute ventilation (VE), heart rate (HR), and arterial oxygen pressure (Pao₂) and arterial carbon dioxide pressure (Paco₂) have not been reported, nor has the relationship of all these parameters to the subjective sensation of respiratory distress been described.

The present report has two purposes: 1) to provide background information concerning these interrelationships, and 2) to determine if data such as vital signs and the sensation or appearance of respiratory distress can eliminate or reduce the need for arterial blood sampling during this period.

*From The Department of Medicine, State University of New York, Upstate Medical Center, Syracuse, N.Y.
**Professor of Medicine.
†Research Technician.
‡Fellow in Pulmonary Diseases.

Supported by USPHS research grant 12995 and Research Career Development Award 1-K3-HE-19414 (Dr. Gilbert) from the National Heart and Lung Institute, Grant PN-10599 from the Heart Association of Upstate New York, The J.A.C. Gray, M.D. Memorial Grant from the New York Tuberculosis and Respiratory Disease Association, Inc., and a grant from the Potts Foundation (Dr. Ashutosh). Manuscript received March 29; revision accepted September 17.

Reprint requests: Dr. Gilbert, Upstate Medical Center, Syracuse 13210

Patients

Fourteen patients were studied. Eight had chronic obstructive pulmonary disease (COPD), and one each had myasthenia gravis, asthma, multiple sclerosis, arteriosclerotic heart disease, traumatic quadriplegia, and carcinoma of the lung. All 14 had been on assisted ventilation from 3 to 80 days, and at the time of study were receiving inspired oxygen concentrations (FIO₂) of 0.21 to 0.40 mm Hg with a volume limited ventilator (Bennett MA-1).

Methods

The measurement of tidal volume by conventional methods requires a mouthpiece and noseclip, or face mask to collect expired air. We have recently shown that this apparatus alters both the tidal volume and respiratory frequency.² In order to avoid these artifacts in the present study, tidal volume off the respirator was obtained with an electromagnetic ventilation monitor (magnetometer) described in detail previously.²-³ Changes in the anterior-posterior dimensions of the chest and abdomen were measured by magnetic coils. The receiver coil for rib cage motion was placed in the midline a variable distance above the xiphoid at a point which gave maximum deflection for quiet breathing; the receiver coil for abdominal motion was placed in the midline 5 cm above the umbilicus. Corresponding exciter coils were placed at the same level on the back. Alternating current sent to the exciter coils produced a magnetic field which induced a voltage in the receiver coils inversely proportional to the distance between the receiver and exciter coils. The signals from the chest and abdomen receiver coils were added electronically to give a total signal representing the tidal volume.

We found these patients unable to perform the breathing maneuvers which in previous studies²-³ were used for the purpose of scaling the chest and abdomen signals. Therefore, the control which adjusted the relative gain of these two signals (ratio control) was arbitrarily set near its midposition...
to give a strong total signal. This total signal was then calibrated with the subject breathing into a spirometer fitted with a calibrated torque potentiometer. He breathed into the spirometer, with no special instructions to alter the breathing pattern. The gain of the total magnetometer signal was then adjusted so that the spirometer and magnetometer signals superimposed. At that point the magnetometer signal had the same calibration as the spirometer. This calibration procedure was repeated at 15-minute intervals during the study; the magnetometer signal was never found to be more than 100 ml different from the spirometer signal once it has been initially set, and usually had not changed at all. This calibration procedure required less than one minute, and the subject was then detached from the spirometer.

Respiratory frequency was obtained from the tidal volume signal with an analog circuit and minute ventilation calculated from VT and f. Tidal volume while on the respirator was obtained from the spirometer in the expiratory line of the respirator. Arterial blood was sampled from an indwelling needle or cannula in the brachial artery and analyzed immediately in duplicate in an analyzer (Instrumentation Laboratories pH/gas). Heart rate and rhythm were obtained from an electrocardiogram monitored throughout the test.

**Experimental Protocol**

Patients were studied at a time when clinical opinion suggested that a trial off the respirator was in order. The experiment started with a 30-minute control period on the respirator at the settings currently in use for clinical management. In all cases the patient triggered the respirator. The patient was then disconnected from the respirator and humidified gas of the same FiO2 as previously supplied by the respirator was delivered to the tracheostomy (nine cases) or endotracheal tube (three cases). In two cases the endotracheal tube was removed when the patient was removed from the respirator; in these cases the appropriate FiO2 was delivered by face mask. Tidal volume, f, and HR were monitored continuously and arterial blood was sampled at 15-minute intervals (sometimes more frequently at the start of the "off" period). The experiment was terminated because of subjective respiratory distress in four patients, and alarming deterioration of PaO2 or Pco2 in two patients. In the remaining eight cases, the patients did not request reconnection to the respirator and the PaO2 and Pco2 levels off the respirator were considered acceptable (PaO2 above 50 mm Hg, Pco2 below 60 mm Hg). In these cases the experiments were terminated (arterial needle and magnetometer removed) after variable periods when the measured parameters seemed stable. The average time off the respirator during the experiment was three hours, with a range of 40 minutes to six and one-half hours.

Since the experiments were performed only when the patients were judged ready for a trial period off the respirator, no ethical considerations were felt to be involved other than those of the patient care situation. During the tests the patients were observed more closely than would have been the case were the experiments not being conducted.

**RESULTS**

**Control Values**

Table 1 shows the average values for f, VT, HR, VE, PaO2, Pco2, and alveolar-arterial oxygen difference for the 14 subjects during the control period on the respirator. FiO2 varied from 0.21 to 0.40; in all cases the automatic cycling was turned off so that the patient controlled the respiratory frequency.

**Table 1**—Control Values for 14 Subjects

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>f</td>
<td>13.1</td>
<td>3.8</td>
<td>7-20</td>
</tr>
<tr>
<td>VT</td>
<td>701</td>
<td>141</td>
<td>550-1100</td>
</tr>
<tr>
<td>VE</td>
<td>9.2</td>
<td>2.4</td>
<td>4.5-11.4</td>
</tr>
<tr>
<td>HR</td>
<td>82</td>
<td>25</td>
<td>48-132</td>
</tr>
<tr>
<td>PaO2</td>
<td>61</td>
<td>8.1</td>
<td>52-76</td>
</tr>
<tr>
<td>Pco2</td>
<td>38</td>
<td>6.9</td>
<td>25-53</td>
</tr>
<tr>
<td>A-a</td>
<td>79</td>
<td>47</td>
<td>42-156</td>
</tr>
<tr>
<td>VC</td>
<td>1014</td>
<td>329</td>
<td>400-1500</td>
</tr>
</tbody>
</table>

f = respiratory frequency, breaths/min
VT = tidal volume, ml
VE = minute ventilation, L/min
HR = heart rate, beats/min
A-a = alveolar-arterial oxygen partial pressure difference calculated from simplified alveolar air equation.
VC = unassisted vital capacity
SD = standard deviation

**Breathing Pattern**

Mean VT fell significantly from a control value of 700 ml to 300 ml and mean f rose significantly from a control value of 13 to 25 breaths per minute as soon

---

**Figure 1.** Mean ± 1 SE for respiratory frequency, tidal volume, and minute ventilation during first hour off respirator for 14 patients. Mean five minute value of VI = 300 ml.
as the patients were disconnected from the respirator (Fig 1) (p < .001 by paired t test). The changes were stable by five minutes, and no further significant changes in mean values occurred after this time. The fall in VT and rise in f occurred in all but one of the 14 patients. Although there were individual changes in VE, the direction of these changes was not uniform, and there was no significant change in mean VE on and off the respirator.

**PaO₂ and PacO₂ Values**

PaO₂ levels fell below the control value in 12 of 14 patients at some time during the period off the respirator; the mean change for all 14 reached statistical significance at 30 minutes (p < .01 by paired t test) at which time mean PaO₂ had fallen from a control value of 61 mm Hg to a value of 54 mm Hg (Fig 2). There were no further significant changes in the mean value afterward.

PaCO₂ levels rose in 11 of the patients following removal from the respirator; the mean change for all 14 attained statistical significance by five minutes (p < .01) at which time mean PacO₂ had risen from a control value of 38 mm Hg to 41 mm Hg. There were no further significant changes in the mean value beyond this time.

Although there were no further significant mean changes in PaO₂ or PacO₂ after 30 minutes, the wide variability of individual changes would preclude statistical significance for the grouped data. In contrast to the changes in VT and f which occurred early and tended to remain relatively fixed during the remainder of the test, individual changes in PaO₂ and PacO₂ occurred in a relatively unpredictable manner throughout the test (for example, see Fig 3, 4).

**Heart Rate and Rhythm**

Heart rate rose an average of 5.4 beats per minute in the first 5 minutes off the respirator (p > .1, not significant), and 9.5 beats per minute by 15 minutes (p < .01). There were no further significant changes during the next 105 minutes. One patient developed ventricular premature beats, which disappeared spontaneously as the test continued. No other arrhythmias occurred during the tests.

**Multiple Sclerosis**

![Graph showing PaO₂ and PacO₂ values](https://www.53702.com/download attachment/paco2.png)
Correlation of PaO₂ and PacO₂ Changes with Changes in VT, f, and HR

PaO₂: A fall in PaO₂ of at least 10 mm Hg from the control value to a level below 50 mm Hg any time during the "off" period was considered physiologically significant. The patients were grouped as to whether they did or did not meet these criteria. The maximum changes in VT, f, VE, HR, and PacO₂ any time during the "off" period as compared to the control period were calculated for each subject. When the changes for the two groups were averaged and compared there was no significant difference for any of the parameters.

PacO₂: A rise in PacO₂ of at least 5 mm Hg from the control value to a level above 45 mm Hg was considered physiologically significant. The subjects were again grouped as to whether they did or did not meet these criteria. There were no statistically significant differences in the maximum changes in VT, f, VE, HR, or PacO₂ between the two groups.

Respiratory Distress

Four patients developed subjective respiratory distress during or shortly after the test while still off the respirator and were placed back on the respirator at their request. Two patients did not complain of distress but were reconnected to the respirator because of alarming deterioration of the blood gas values (PaO₂ of 40, PacO₂ of 38 in one case, PaO₂ of 46, PacO₂ of 64 in the other). These six patients were compared to the eight patients who had completely successful trial periods off the respirator. There was no difference in the control values for unassisted vital capacity or f in the two groups. Although the control value for mean PaO₂ was similar for the two groups, the mean control value for alveolar-arterial oxygen difference was actually wider in the group who did not require reconnection to the respirator (p < .05). This finding was due to the higher FiO₂ values required in these subjects to achieve the same control values for PaO₂ as the subjects who did require reconnection to the respirator, indicating a greater defect in gas exchange in the former group. The maximum changes in f, VT, VE, HR, and PaO₂ were not significantly different in the two groups during the "off" period compared to the control period. If the two subjects who did not develop respiratory distress but who were reconnected to the respirator because of apprehension on the part of the investigators because of deterioration of PaO₂ or PacO₂ levels are excluded, the results of the analysis are not changed.

The three patients who were being monitored when they developed respiratory distress showed a fall in PaO₂ and a rise in PacO₂ values at that time. Five patients, however, showed changes of a similar degree and to a comparable absolute level, without complaining of respiratory distress.

Comparison of COPD and Non-COPD Patients

The data were examined to see if the responses in the eight patients with COPD were different from those in the six patients without COPD. There was no significant difference in the percentage of successful vs unsuccessful trials off the respirator in the two groups, nor in the maximum changes in f, VT, VE, HR, PaO₂ or PacO₂.

Table 2 lists the mean values for the maximum changes in the various parameters with the patients grouped according to whether they did or did not develop hypoxemia, hypercapnia, or respiratory distress, or whether or not the primary diagnosis was COPD.

Correlation of Changes in f and VT

Since respiratory frequency is so much easier to measure than is tidal volume in the patient not on a
Table 2—Mean Changes in Respiratory Parameters*

<table>
<thead>
<tr>
<th></th>
<th>Hypoxemia</th>
<th></th>
<th>Hypercapnia</th>
<th></th>
<th>Resp. distress</th>
<th></th>
<th>COPD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>VT</td>
<td>434</td>
<td>385</td>
<td>379</td>
<td>465</td>
<td>408</td>
<td>411</td>
<td>387</td>
</tr>
<tr>
<td>f</td>
<td>13.2</td>
<td>14.4</td>
<td>14.8</td>
<td>12.2</td>
<td>12.3</td>
<td>15.0</td>
<td>13.4</td>
</tr>
<tr>
<td>VE</td>
<td>1.1</td>
<td>2.0</td>
<td>2.1</td>
<td>0.4</td>
<td>0.8</td>
<td>2.0</td>
<td>2.1</td>
</tr>
<tr>
<td>HR</td>
<td>18.8</td>
<td>18.3</td>
<td>14.8</td>
<td>21.0</td>
<td>17.2</td>
<td>14.6</td>
<td>19.1</td>
</tr>
<tr>
<td>Pa02</td>
<td>—</td>
<td>—</td>
<td>-9.7</td>
<td>-9.8</td>
<td>-10.2</td>
<td>-9.4</td>
<td>-10.2</td>
</tr>
<tr>
<td>PaCO2</td>
<td>5.4</td>
<td>8.4</td>
<td>—</td>
<td>—</td>
<td>8.7</td>
<td>5.6</td>
<td>6.9</td>
</tr>
</tbody>
</table>

* Symbols as in Table 1.

respirator, we were interested to see if changes in VT could be predicted from changes in f. The large decreases in VT which occurred immediately upon removal from the respirator were always accompanied by an increase in f. The magnitude of the changes in VT, however, did not correlate with the magnitude of the changes in f. In order to see if smaller changes in VT were accompanied by reciprocal changes in f, consecutive recorded values for VT and f, usually 15 minutes apart, were examined for all 14 patients for the period off the respirator. A change of 100 ml or more in VT between consecutive values was investigated. Nine such examples were found; in seven there was a reciprocal change in f. In two of these seven, however, the change in f was only one breath per minute. We conclude that under these circumstances, large decreases in VT (250 ml or more) will be accompanied by increases in f, but smaller changes may be missed.

**DISCUSSION**

The values of VT on the respirator (550-1,100 ml) for the patients in this study are in the range shown to be necessary for patients in respiratory failure. The directional changes in VT and f on removal from the respirator are not surprising since the control values for VT are generally higher and the control values for f generally lower than those seen in normal subjects and patients with pulmonary disease during quiet, unassisted breathing. The tidal volumes on the respirator were undoubtedly too large for these seriously ill patients to sustain unassisted; the fall in VT represents an adjustment to a more realistic breathing pattern for these conditions.

In contrast to the predictable and immediate changes in the breathing pattern, the changes in Pa02 and PaCO2 were variable. The presence or absence of progressive hypoxemia or hypercapnia could not be inferred from the changes in VT, f, VE, HR, nor from the patient's subjective response. The patient with multiple sclerosis (Fig 3) illustrates this dissociation. On withdrawal from the respirator there was an immediate rise in HR and f, a fall in VT, and a progressive fall in Pa02 levels. PaCO2 values did not change. After 75 minutes Pa02 levels had decreased to 40 mm Hg, and the patient was started on 50 percent oxygen by a tracheal collar. Pa02 values rose to 63 mm Hg with no change in HR, VT, or PaCO2 and with a further rise in f. During the entire test the patient never complained of respiratory distress.

Two patients maintained surprisingly good values for Pa02 and PaCO2 despite what would ordinarily be considered a very unfavorable breathing pattern off the respirator. The respiratory data of one of these patients are shown in Figure 4. Immediately following removal from the respirator, VT fell from 1,100 to 300 ml, f rose from 9 to 20 breaths per minute and VE fell from 10 to 6 liters per minute. These values remained relatively stable over the next hour, while the Pa02 value rose progressively. We interpret this improvement in Pa02 level despite a fall in VE with a relative increase in anatomic deadspace ventilation as evidence for a more uniform distribution of ventilation relative to perfusion off the respirator compared to on the respirator. An alternative explanation would be a reduction in oxygen uptake off the respirator; this measurement was not made, but since one of the purposes of assisted ventilation is to lower the work of breathing, an appreciable fall in oxygen uptake off the respirator seems unlikely.

Should the Pa02 and PaCO2 levels be the final criteria for judging the success or failure of the trial period off the respirator? The body adapts surprisingly well to chronic hypoxemia, and extremely low values of Pa02 have been reported with ultimate survival. Several of our subjects developed values of Pa02 below 45 mm Hg without other manifestations of lack of oxygen such as sweating, restlessness, respiratory distress, tachycardia, or significant cardiac arrhythmias, suggesting that this hypoxemia may not have been accompanied by tissue hypoxia of a critical degree. Because of the steep slope of the oxygen dissociation curve below 50 mm Hg, how-

---

**Table 2—Mean Changes in Respiratory Parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Yes</th>
<th>No</th>
<th>Yes</th>
<th>No</th>
<th>Yes</th>
<th>No</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>f</td>
<td>13.2</td>
<td>14.4</td>
<td>14.8</td>
<td>12.2</td>
<td>12.3</td>
<td>15.0</td>
<td>13.4</td>
<td>14.5</td>
</tr>
<tr>
<td>VE</td>
<td>1.1</td>
<td>2.0</td>
<td>2.1</td>
<td>0.4</td>
<td>0.8</td>
<td>2.0</td>
<td>2.1</td>
<td>0.7</td>
</tr>
<tr>
<td>HR</td>
<td>18.8</td>
<td>18.3</td>
<td>14.8</td>
<td>21.0</td>
<td>17.2</td>
<td>14.6</td>
<td>19.1</td>
<td>11.1</td>
</tr>
<tr>
<td>Pa02</td>
<td>—</td>
<td>—</td>
<td>-9.7</td>
<td>-9.8</td>
<td>-10.2</td>
<td>-9.4</td>
<td>-10.2</td>
<td>-9.0</td>
</tr>
<tr>
<td>PaCO2</td>
<td>5.4</td>
<td>8.4</td>
<td>—</td>
<td>—</td>
<td>8.7</td>
<td>5.6</td>
<td>6.9</td>
<td>7.0</td>
</tr>
</tbody>
</table>
ever, it seems prudent not to allow an acutely ill patient to have a lower value of \( \text{Pao}_2 \) for any sustained period.6

The present study provides background information useful in assessing the trial off the respirator. An immediate rise in \( f \) and fall in VT can be anticipated and does not necessarily indicate that the patient will soon develop intolerable respiratory distress or critical deterioration of \( \text{Pao}_2 \) or \( \text{Paco}_2 \). A rise in heart rate or the appearance of arrhythmias should certainly be cause for concern, but significant deterioration can occur in \( \text{Pao}_2 \) and \( \text{Paco}_2 \) values without these changes, and without changes in the general appearance of the patient or the development of the subjective sensation of dyspnea. If it is considered vital that the \( \text{Pao}_2 \) level be kept above 50 mm Hg, at present noninvasive monitoring cannot be said to provide the necessary information.

ACKNOWLEDGMENT: We gratefully acknowledge the technical assistance of Mrs. Jane Bowman and Mr. Thomas Birkel.

REFERENCES