Oxygen Cost of Breathing in Postoperative Patients*

Pressure Support Ventilation vs Continuous Positive Airway Pressure

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In seven postoperative patients with normal preoperative pulmonary function tests, we evaluated the oxygen cost of breathing (\(\dot{V}_O_2\),resp) during continuous positive pressure ventilation (CPAP) and during a 15 cm H\(_2\)O inspiratory pressure support ventilation (IPSV). For both periods, \(\dot{V}_O_2\) resp was estimated as the difference between total oxygen uptake of the period (\(\dot{V}_O_2\)tot), measured by a mass-spectrometer system, and that during controlled ventilation. During CPAP ventilation, \(\dot{V}_O_2\) resp was found to be 11.2 ± 1.4 percent of \(\dot{V}_O_2\)tot. During IPSV, \(\dot{V}_O_2\) resp was found insignificant. It is concluded that a 15 cm H\(_2\)O IPSV takes over the major part of the work of breathing in postoperative patients without preexisting pulmonary disease.

During weaning from artificial ventilation, changing from a controlled to a spontaneous mode of respiration is associated with an increase in whole-body oxygen consumption (\(\dot{V}_O_2\)tot). This increase, called oxygen cost of breathing (\(\dot{V}_O_2\)resp), is less than 5 percent of \(\dot{V}_O_2\)tot in normal subjects breathing quietly.\(^1\) In critically ill or postoperative patients, whose tracheas were intubated, \(\dot{V}_O_2\) resp has been found to be in the order of 20 percent of \(\dot{V}_O_2\)tot.\(^2,3\)

Recently, inspiratory pressure support ventilation (IPSV) has been proposed to decrease the work of breathing in spontaneously breathing patients.\(^4\) We hypothesized that IPSV can reduce significantly \(\dot{V}_O_2\) resp in the weaning period of artificial ventilation. We therefore investigated \(\dot{V}_O_2\) resp in postoperative patients breathing alternatively with IPSV and with continuous positive pressure ventilation (CPAP).

Materials and Methods

We studied seven tracheally intubated male patients who required a two to three days of respiratory support after a major surgical procedure. They had normal results preoperative pulmonary function tests. Ventilation was provided with a ventilator (Siemens Servo C). Clinical data are given in Table 1.

All patients were studied on the morning preceding extubation. At this time, they could alternately be breathing spontaneously, or be on controlled ventilation. They were normoventolic and had normal cardiovascular functions. The nutritional intake was kept constant throughout the study.

Protocol

The protocol was approved by the ethical committee of our institution, and informed consent was obtained from the patients' nearest relative.

Three consecutive 60-minute study periods were achieved, according to a randomized order: controlled ventilation (CV), CPAP, and IPSV. For the three ventilatory modes, the same low level of PEEP (5 cm H\(_2\)O) was applied to the patients in order to diminish the incidence of postoperative respiratory complications.\(^5\) During CV, the ventilator frequency and minute volume were adjusted so that the patients appeared relaxed and comfortable and did not trigger inflation or resist gas flow. During IPSV, pressure support was set at 15 cm H\(_2\)O, and the trigger level at −2 cm H\(_2\)O.

During the second half of each period, \(\dot{V}_O_2\) tot, carbon dioxide production (\(\dot{V}_C0_2\)), respiratory quotient (RQ), tidal volume (\(V_t\)), respiratory rate (RR), and minute volume (\(V_e\)) were continuously measured and recorded at three-minute intervals. At the conclusion of each period, an arterial blood sample was taken for the measurements of \(P_{O_2}\), \(P_{C0_2}\), and \(H^+\) concentration. In one patient who had no esophageal surgery (patient 7), simultaneous recordings of respiratory flow, airway pressure, esophageal pressure, and gastric pressure were achieved during the application of the three ventilatory modes.

Measurements and Calculations

\(P_{O_2}\), \(P_{C0_2}\) and \(H^+\) concentration were measured using standard electrodes. For the measurements of pressures and flow in patient 7, we used pressure transducers, a pneumotachograph (Gould), and a four-channel recorder (Gould). Esophageal and gastric pressures were measured by two balloon-catheters joined to each other. The esophageal balloon was positioned 10 cm above the cardia. The balloons were 5 cm long and were filled with 0.5 ml air. Pulmonary gas exchange was measured with our mass-spectrometer-microcomputer system. Details of this procedure and thorough validation have been given in a previous report.\(^6\) The system can be briefly described as follows. Gas samples were drawn from the Y piece of the patient's breathing circuit to the mass-spectrometer and analyzed for inspired \(O_2\) concentration and \(C0_2\) waveform recognition. The latter analysis allowed rejection of artifacted cycles, such as coughing. Then, expired gas was sampled from the outlet of a mixing chamber for the measurement of mixed expired \(O_2\) and \(C0_2\) concentrations. Expired flow was measured by a pneumotachograph (Gould). All the signals were collected by the microcomputer which was programmed to reject artifacted respiratory sequences and to compute \(V_t\), RR, \(V_e\),

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Manuscript received April 30; revision accepted August 3.

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Table 1—Patients' Characteristics Upon Entry Into the Study

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age</th>
<th>Diagnosis and Surgical Procedure</th>
<th>Peak Pw, cm H2O</th>
<th>FIO2</th>
<th>Internal diameter of endotracheal tube (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>59</td>
<td>Esophagus carcinoma: cervicotomy + thoracotomy + laparotomy</td>
<td>38</td>
<td>0.35</td>
<td>8.5</td>
</tr>
<tr>
<td>2</td>
<td>58</td>
<td>Cardia carcinoma: thoracotomy + laparotomy</td>
<td>32</td>
<td>0.34</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>49</td>
<td>Esophagus carcinoma: thoracotomy + laparotomy</td>
<td>30</td>
<td>0.33</td>
<td>7.5</td>
</tr>
<tr>
<td>4</td>
<td>57</td>
<td>Esophagus carcinoma: thoracotomy + laparotomy</td>
<td>32</td>
<td>0.36</td>
<td>8</td>
</tr>
<tr>
<td>5</td>
<td>53</td>
<td>Esophagus carcinoma: thoracotomy + laparotomy + cervicotomy</td>
<td>42</td>
<td>0.37</td>
<td>8</td>
</tr>
<tr>
<td>6</td>
<td>55</td>
<td>Esophagus carcinoma: cervicotomy + laparotomy</td>
<td>38</td>
<td>0.39</td>
<td>8</td>
</tr>
<tr>
<td>7</td>
<td>42</td>
<td>Peritonitis: laparotomy</td>
<td>36</td>
<td>0.31</td>
<td>8</td>
</tr>
</tbody>
</table>

*Peak Pw, value of the peak airway pressure under controlled ventilation.

V\textsubscript{O}, \textsubscript{VCO}, and RQ at three-minute intervals. For each study period, the mean values of all the measured parameters were calculated. During each period of spontaneous respiration (CPAP and IPSV), \textsubscript{O} resp was calculated as the difference between \textsubscript{O}tot of the period, and \textsubscript{O}tot measured during CV. Results are presented as the mean ± SE and further statistical analysis used two-way analysis of variance with Duncan's multiple range follow-up tests.

RESULTS

As indicated in Table 2, CV was associated with a slight respiratory alkalosis, which could be accounted for by \textsubscript{V}r and \textsubscript{V}E values higher than that observed during CPAP and IPSV. There was no difference for \textsubscript{PaO}2. The \textsubscript{V}E was the same for the two modes of spontaneous respiration, but RR was more elevated during CPAP. Figure 1 shows that CPAP resulted in all patients having a significant increase in \textsubscript{O}2 tot. The calculated \textsubscript{O}2 resp was 11.2 ± 1.4 percent of \textsubscript{O}2 tot (range 6.6 to 15.6 percent), or 2.6 ± 0.4 ml O2/L of ventilation (range 1.7 to 4.2 ml O2/L of ventilation).

Conversely, there was no difference for \textsubscript{V}O2 tot between IPSV and CV. Blood pressure and heart rate remained stable throughout the study. Figure 2 shows the recordings of pressures and flow in the patient 7. During inspiration with IPSV, esophageal pressure decreased, whereas gastric pressure increased, thus indicating a sustained diaphragmatic contraction.

DISCUSSION

In the present study, \textsubscript{O} resp was estimated as the difference between \textsubscript{O}tot during spontaneous respiration and that during controlled ventilation. Two points should be emphasized. First, for the estimation of \textsubscript{O} resp, we used a mass spectrometer system which has the advantage of giving an accurate continuous measurement of \textsubscript{O}, in contrast with the Fick method, or the Douglas bag method which have the limitation of intermittent data availability. Secondly, during CV, particular attention was paid to ensure that ventilation was entirely passive, without any inspiratory, or expiratory effort. To obtain this complete relaxation, it was most often necessary to induce a certain degree of hyperventilation, leading to a decrease in H concentrations. It has been shown that \textsubscript{O}, is pH dependent, and that respiratory alkalosis is associated with an increase in \textsubscript{O}. So, in our study, \textsubscript{O} might have been slightly over-estimated during CV, and thus, \textsubscript{O} resp might have been slightly underestimated. The hyperventilation associated with CV may also explain the

Table 2—Mean Values of Physiologic Parameters for Three Ventilatory Conditions

<table>
<thead>
<tr>
<th></th>
<th>CV</th>
<th>CPAP</th>
<th>IPSV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vr (ml)</td>
<td>746 ± 62</td>
<td>480 ± 43*</td>
<td>639 ± 47†</td>
</tr>
<tr>
<td>Respiratory rate (min⁻¹)</td>
<td>18 ± 1</td>
<td>23 ± 1*</td>
<td>16 ± 1†</td>
</tr>
<tr>
<td>V (L·min⁻¹)</td>
<td>13.2 ± 0.6</td>
<td>10.7 ± 0.7*</td>
<td>10.3 ± 0.5*</td>
</tr>
<tr>
<td>PaO2 (mm Hg)</td>
<td>89.3 ± 8.5</td>
<td>105 ± 10.4</td>
<td>94.5 ± 6.1</td>
</tr>
<tr>
<td>PaCO2 (mm Hg)</td>
<td>27.3 ± 1.1</td>
<td>34 ± 1.8*</td>
<td>33.1 ± 1.8*</td>
</tr>
<tr>
<td>H⁺ concentration (nMoles·L⁻¹)</td>
<td>28.7 ± 1.6</td>
<td>94.1 ± 1.2*</td>
<td>33.8 ± 0.9*</td>
</tr>
<tr>
<td>\textsubscript{V}O2 tot (mL·min⁻¹·m⁻²)</td>
<td>141 ± 7</td>
<td>157 ± 9*</td>
<td>140 ± 7†</td>
</tr>
<tr>
<td>\textsubscript{V}O2 resp (mL·min⁻¹·m⁻²)</td>
<td>...</td>
<td>16.2 ± 2.9</td>
<td>-0.3 ± 1.2†</td>
</tr>
<tr>
<td>\textsubscript{V}CO2 (mL·min⁻¹·m⁻²)</td>
<td>129 ± 5</td>
<td>135 ± 6*</td>
<td>125 ± 5†</td>
</tr>
<tr>
<td>RQ</td>
<td>0.91 ± 0.01</td>
<td>0.86 ± 0.01*</td>
<td>0.89 ± 0.02</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>132 ± 3</td>
<td>139 ± 5</td>
<td>133 ± 5</td>
</tr>
<tr>
<td>Heart rate (min⁻¹)</td>
<td>91 ± 6</td>
<td>93 ± 6</td>
<td>88 ± 5</td>
</tr>
</tbody>
</table>

*Significant by comparison with CV period (p<0.05).
†Significant for the comparison between CPAP and IPSV (p<0.05).
higher value of RQ which was observed during this period. Indeed, 30 minutes after the onset of CV, the CO₂ washout was probably not completed, leading to an overestimation of VCO₂ and thus, of RQ.¹⁰

The values of \( \dot{V}O_2 \) resp which we measured in our patients during CPAP ventilation were higher than those measured in normal subjects at rest.¹¹ Several factors can explain this result. The stress of spontaneous breathing, with an increase in catecholamine release, might be a first contributor. We did not measure plasma catecholamines, but it must be noted that heart rate and systolic blood pressure remained stable when the ventilatory conditions were changed. Probably, a more significant factor is dealing with the work of breathing. In the postoperative period of abdominal surgery, there is a slight shift from predominantly abdominal breathing to rib cage breathing.¹² This change may induce an increase in respiratory work and a change in the mechanical efficiency of the respiratory muscles. Finally, there is additional inspiratory work of breathing during CPAP ventilation to trigger the ventilator and to overcome the resistances due to an eventual imbalance between the patient's inspiratory demand and the inspiratory flow delivered by the breathing apparatus.¹³,¹⁴

Our results are close to those published by Thung et al.¹⁵ These authors found \( \dot{V}O_2 \)resp to be 13 percent of \( \dot{V}O_2 \)tot in patients studied after cardiac surgery. Higher values have been reported by Wilson et al.¹⁶ in the immediate postoperative period of cardiac surgery. In patients with various degrees of cardiorespiratory diseases, \( \dot{V}O_2 \) resp was found three times higher than that reported in the present study (⁸.7 vs 2.6 ml O₂/L of ventilation). This is related to the underlying lung disease which is associated with an increased work of breathing and/or a decreased efficiency of the respiratory muscles.

Compared to CPAP ventilation, IPSV was associated with a lower RR and a higher TV. Similar changes have been observed by other investigators.¹⁷ The normal PaCO₂ values which we found during IPSV confirms that this mode of ventilation allows the patients to match their minute ventilation to their \( \dot{V}CO_2 \).¹⁸ During IPSV, the level of pressure support was set at 15 cm H₂O, a value lower than the 30 to 42 cm H₂O peak airway pressures which were observed during CV. This suggests that, under IPSV, an active inspiratory effort was still required by the patients to provide a tidal volume of the same order as the one observed during controlled ventilation. This assumption is also supported by the sustained inspiratory diaphragmatic contraction which was observed in patient 7 during the application of IPSV. However, we found that the amount of oxygen required to supply energy to the respiratory muscles under IPSV was insignificant, since it did not affect whole body oxygen consumption. In the same way, it can be assumed from our data that the work which is necessary at the onset of inspiration to trigger the ventilator is of little magnitude.

In conclusion, the present study was conducted in the weaning period of a major surgical procedure in seven patients with normal preoperative pulmonary function test results. We found \( \dot{V}O_2 \)resp to be about 11 percent of \( \dot{V}O_2 \)tot. Secondly, under IPSV, \( \dot{V}O_2 \)resp was insignificant, which indicates that this mode of ventilation took over the major part of these patients'...
work of breathing.

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