Changes in occlusion pressure ($P_{0.1}$) and breathing pattern during pressure support ventilation

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Abstract

Background—The purpose of this study was to investigate changes in breathing pattern, neuromuscular drive ($P_{0.1}$), and activity of the sternocleidomastoid muscles (SCM) during a gradual reduction in pressure support ventilation (PSV) in patients being weaned off controlled mechanical ventilation.

Methods—Eight non-COPD patients recovering from acute respiratory failure were included in this prospective interventional study. All patients were unable to tolerate discontinuation from mechanical ventilation. Each patient was evaluated during a period of spontaneous breathing and during PSV. Four successive levels of PSV were assessed in the following order: 20 cm H$_2$O (PS20), 15 cm H$_2$O (PS15), 10 cm H$_2$O (PS10), and 5 cm H$_2$O (PS5).

Results—When pressure support was reduced from PS20 to PS10 the respiratory rate ($f$) and the rapid shallow breathing index ($f/VT$) significantly increased and tidal volume ($VT$) significantly decreased. These parameters did not vary when pressure support was reduced from PS10 to PS5. Conversely, $P_{0.1}$ values between PS20 and PS15 but increased significantly at low PSV levels. $P_{0.1}$ values were always greater than 2.9 cm H$_2$O (4.1 (1.1) cm H$_2$O) when SCM activity was present. When contraction of the SCM muscles reappeared the $P_{0.1}$ was the only parameter that changed significantly.

Conclusions—In postoperative septic patients the value of $P_{0.1}$ seems to be more applicable as an index of the neuromuscular drive during PSV. $P_{0.1}$ values differentiate between those patients who can be successfully weaned from mechanical ventilation and those who cannot.

Measurement of $P_{0.1}$, the negative airway pressure generated during the first 100 ms of an occluded inspiration, was introduced by Whitelaw et al.11 Because it is measured at zero flow and is independent of respiratory system compliance and resistance, it is an estimate of the neuromuscular drive to breathe. High $P_{0.1}$ values reflect an increased neuromuscular activation of the respiratory system and indicate a strong likelihood of inspiratory muscle fatigue. $P_{0.1}$ has also been shown to be closely correlated with the work of breathing in patients receiving PSV.9 It was recently suggested that $P_{0.1}$ is a better index than breathing frequency for estimating the change in load for the respiratory muscles.10

Few studies have described both the breathing pattern parameters and changes in $P_{0.1}$ during variation of PSV levels.3,15 Although they reported an increase in $P_{0.1}$ values with the decrease in PSV levels, Alberti and coworkers10 showed a greater sensitivity of this index for high PSV levels whereas Berger and associates10 found a greater sensitivity for low PSV levels.

The objective of this study was to assess the changes in breathing pattern parameters and neuromuscular drive during gradual reduction of the PSV level in patients being weaned from mechanical ventilation and to determine whether a $P_{0.1}$ threshold exists when SCM contractions appear.

Methods

Eight patients recovering from acute respiratory failure of various causes were studied prospectively following approval by the institutional ethics committee of the Hospital St Eloi and Montpellier. 34295 Cedex, France. Montpellier, France.
ethics committee. All patients had undergone continuous mechanical ventilation for more than 48 hours. At the beginning of the study no patient had an internal positive end expiratory pressure (iPEEP) as measured during continuous mechanical ventilation by an end expiratory occlusion manoeuvre obtained by depression of the expiratory pause button on the Servo Ventilator 900C (Siemens, Berlin, Germany). The clinical and respiratory characteristics of the patients are shown in Table 1. All patients were considered to be ready for weaning off ventilation by the following criteria: partial or total recovery from their underlying condition, body temperature <38.5°C, no evidence of infection, satisfactory renal function, neuropsychological state compatible with autonomous respiration, correct metabolic equilibrium, haemoglobin level >8 g/dl, absence of clinical signs of left ventricular dysfunction, and no cardiac rhythm or conduction disturbances. All sedative drugs, hypnotics, and narcotics were withheld for 24 hours prior to the weaning trial.

All weaning trials were performed in the morning. Patients were maintained in a semi-recumbent position. They underwent a 20 minute test period of spontaneous T-piece breathing with an FIO2 of 40%. All spirometric, gas exchange, and pressure measurements were obtained during the last 10 minutes of each trial. All recordings and measurements were obtained during the last 10 minutes of each trial in the following order: breathing pattern, patellar reflex, and a model CD15 carrier demodulator.

Table 1 Clinical and respiratory characteristics of the patients during spontaneous breathing

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (years)</th>
<th>PaO2 (mm Hg)</th>
<th>PaCO2 (mm Hg)</th>
<th>pH</th>
<th>Pmax (cm H2O)</th>
<th>P0.1/Pmax</th>
<th>CMV duration (days)</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>67</td>
<td>70</td>
<td>38</td>
<td>7.49</td>
<td>60</td>
<td>0.1</td>
<td>3</td>
<td>Postoperative sepsis (colonic surgery)</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>73</td>
<td>73</td>
<td>33</td>
<td>7.33</td>
<td>44</td>
<td>0.13</td>
<td>20</td>
<td>Postoperative sepsis (coronary bypass)</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>89</td>
<td>96</td>
<td>35</td>
<td>7.49</td>
<td>25</td>
<td>0.25</td>
<td>18</td>
<td>ARDS (pneumonia)</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>69</td>
<td>72</td>
<td>37</td>
<td>7.49</td>
<td>69</td>
<td>0.09</td>
<td>21</td>
<td>Postoperative sepsis (coronary bypass)</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>49</td>
<td>112</td>
<td>36</td>
<td>7.38</td>
<td>32</td>
<td>0.185</td>
<td>11</td>
<td>Postoperative sepsis (gastroscopy)</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>78</td>
<td>74</td>
<td>60</td>
<td>7.39</td>
<td>50</td>
<td>0.055</td>
<td>6</td>
<td>Postoperative sepsis (abdominal aortic surgery)</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>65</td>
<td>101</td>
<td>34</td>
<td>7.43</td>
<td>45</td>
<td>0.108</td>
<td>16</td>
<td>Postoperative sepsis (abdominal aortic surgery)</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>70</td>
<td>67</td>
<td>51</td>
<td>7.42</td>
<td>52</td>
<td>0.29</td>
<td>18</td>
<td>Postoperative sepsis (oesophagectomy)</td>
</tr>
</tbody>
</table>

PaO2, PaCO2 = arterial oxygen and carbon dioxide tensions; Pmax = maximal inspiratory pressure; P0.1 = occlusion pressure; CMV = continuous mechanical ventilation; ARDS = adult respiratory distress syndrome.

Table 2 Variation in breathing pattern and occlusion pressure (P0.1) with different levels of pressure support (PS) ventilation

<table>
<thead>
<tr>
<th>PS20</th>
<th>PS15</th>
<th>PS10</th>
<th>PS5</th>
<th>SB</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>f (breaths/min)</td>
<td>VE (l/min)</td>
<td>Vt (l)</td>
<td>VT/Ti (s)</td>
<td>Ti/Ttot</td>
<td>f/VT (breaths/min)</td>
</tr>
<tr>
<td>16 (4.3)</td>
<td>11.2 (2.1)</td>
<td>0.76 (0.15)</td>
<td>0.62 (0.21)</td>
<td>0.35 (0.06)</td>
<td>21.9 (6.7)</td>
</tr>
<tr>
<td>21 to 27</td>
<td>8.5 to 15.8</td>
<td>0.41 to 0.61</td>
<td>0.40 to 0.70</td>
<td>0.32 to 0.40</td>
<td>34 to 68</td>
</tr>
<tr>
<td>24 to 33</td>
<td>10.1 to 14.1</td>
<td>0.36 to 0.50</td>
<td>0.41 to 0.61</td>
<td>0.36 to 0.42</td>
<td>50 to 89</td>
</tr>
<tr>
<td>26 to 36</td>
<td>8.2 to 13</td>
<td>0.27 to 0.41</td>
<td>0.32 to 0.51</td>
<td>0.37 to 0.46</td>
<td>68 to 122</td>
</tr>
</tbody>
</table>

Values are mean (SD) with 95% confidence intervals.
f = respiratory rate; VE = minute volume; Vt = tidal volume; VT/Ti = mean inspiratory flow; Ti/Ttot = ratio of inspiratory to total time of the respiratory cycle; f/VT = rapid shallow breathing index; P0.1 = occlusion pressure; SB = spontaneous breathing.

MEASUREMENTS
Breathing pattern parameters were recorded for one minute using an electronic spirometer (Ultima SV, Datex, Helsinki, Finland) and the following measurements were made: VT, f, Vt, inspiratory time (Ti), total time of the respiratory cycle (Ttot), ratio of inspiratory to total time of the respiratory cycle (Vi/Ttot), and the rapid shallow breathing index (f/VT). Airway pressure was recorded at the proximal end of the endotracheal tube with a differential pressure transducer ±50 cm H2O (Validyne MP45) and a model CD15 carrier demodulator.
P0.1 measurement during spontaneous breathing (T-piece)
The P0.1 was measured using a one way silent manually activated valve that was occluded at the end of expiration by means of a syringe. The patient saw neither the valve nor the operator, and thus could not anticipate the occlusion that lasted, on average, less than 300 ms.

P0.1 measurement during PSV
The P0.1 was measured using an end expiratory occlusion manoeuvre obtained by depressing the expiratory pause button on the Servo Ventilator 900C.16 The inspiratory scissors valve remained closed at the end of expiration and the flap valve closed on the expiratory side, resulting in inspiratory effort against a closed system. Once the initial inspiratory effort was completed, the inspiratory button was released and normal respirations resumed.

Maximal inspiratory pressure (PImax)
The technique of Marini et al17 was employed, using an unoccluded exhalation circuit, to measure PImax with a differential pressure transducer ±150 cm H2O (Newark Electronics, USA). A minimum of three measurements were performed. The P0.1/PImax ratio was then calculated. All signals were amplified, recorded and printed on a Gould Windograf recorder. P0.1 and PImax measurements were made at paper speeds of 50 mm/s and 10 mm/s, respectively. A minimum of 3–5 reproducible measurements were performed for P0.1, each separated by the time needed for a return to resting ventilation levels. Instruments were calibrated before each procedure with two water manometers.

Two physicians independently assessed activity of the SCM muscles by palpating the muscles in the neck. SCM contraction was considered to be present if there was agreement between the two physicians regarding the presence of SCM activity. The optimal level of PSV was defined by the lowest PSV level without SCM contraction.

The following parameters were continuously monitored: cardiac frequency, oscillometrically measured systolic blood pressure, mean and diastolic arterial blood pressure, end tidal CO2, and pulse oximetry (Ultima SV, Datex, Helsinki, Finland). Arterial blood gases were sampled during mechanical ventilation and immediately following the spontaneous T-piece breathing trial via an arterial catheter.

The physician terminated the trial if a patient had any of the following signs of poor tolerance: f >35 breaths/min, SaO2 <90%, heart rate >140 beats/min, systolic blood pressure >180 mm Hg or <90 mm Hg, agitation, or anxiety.

STATISTICAL ANALYSIS
Results are expressed as mean (SD) values with 95% confidence intervals. The ANOVA test for repeated measurements was simultaneously applied to all five treatments and the comparison test used was the PLSD of Fisher. The Wilcoxon test for small samples was used to compare quantitative variables when activity of the SCM muscles was present. A p value of <0.05 was considered significant.

Results
The breathing pattern and occlusion pressure data are summarised in table 2. When pressure...
and PSV, and his P0.1 was lower than seen in patient 6 during spontaneous breathing.

**Discussion**

In postoperative septic patients we have shown that the value of P0.1 as measured using the technique of Brenner et al., appears more useful than breathing pattern parameters for setting the optimal level of pressure assistance during PSV. When SCM muscle contraction occurred it was the only parameter significantly modified.

Several authors have found that VT varies directly, and f and f/Vt inversely, with the level of PSV. Thus, when muscles are almost unloaded (at high levels of PSV) the patient breathes with high VT and low frequency. VT, Ti, and f did not change significantly at low levels of PSV10 to PS5. VE, VT/Ti, and Ti/Ttot did not vary significantly when the PSV level was reduced.

Contraction of the SCM muscles occurred in seven patients during spontaneous breathing and at PS5, but only in patient 8 at PS10 (table 3). Only P0.1 was modified when SCM muscle activity was present (fig 1; p<0.05). When there was no SCM muscle activity P0.1 values were always lower than 2.9 cm H2O (1.8 (0.5) cm H2O). No activity of the SCM muscles was seen in patient 6 during spontaneous breathing and PSV, and his P0.1 was lower than 2.8 cm H2O at each stage. Individual values of P0.1 are shown in fig 2.

All patients remained stable with no signs of poor tolerance during the trial.

During weaning off continuous mechanical ventilation it is difficult to determine the optimal level of PSV as defined by the maintenance of diaphragm activity and the avoidance of diaphragmatic fatigue. Specific indexes of inspiratory muscle fatigue are only obtained using invasive techniques. However, in the study by Brochard et al., contraction of SCM muscles was evaluated at the bedside and appeared at the same time as diaphragmatic fatigue, as evaluated by electromyography. In the present study none of the breathing pattern parameters was significantly modified when contraction of SCM muscles reappeared. Other authors have found that, for an “acceptable breathing pattern” with f between 15 and 25 breaths/min, the respiratory muscle work-load can be excessive leading to fatigue and predisposing to muscle atrophy. Conversely, persistent tachypnoea (range 22–38 breaths/min) occurred in the absence of patient work of breathing. Similarly, Alberti et al. found no correlation between breathing pattern parameters and work of breathing. This suggests that breathing pattern parameters are not an accurate assessment of the optimal threshold of PSV.

Occlusion pressure has been shown to be a predictor of the success of weaning in patients with or without obstructive lung disease. High levels of P0.1 are associated with increased respiratory effort and indicate an inability to breath independently with success. In contrast, lower values of P0.1 are associated with effective weaning. Moreover, Murciano et al. have shown that during weaning trials a good relationship exists between P0.1 and the high to low ratio of the diaphragmatic electromyogram. In our study P0.1 varied inversely with the level of PSV. More interesting was the fact that P0.1 did not change significantly with high levels of PSV. Similarly, Berger et al. found that P0.1 did not vary with further increases in PSV to levels above the crossover to total unloading. At these high levels we assume that patients were almost completely unloaded and the only work of breathing was to trigger the ventilator. Conversely, P0.1 changed significantly with lower PSV levels when contraction of the SCM muscles occurred. At this time inspiratory effort increases in order to keep alveolar ventilation in an acceptable range. Thus, an acute increase in the P0.1 value may signal an insufficient PSV level. In a recent study Lotti et al. found a significant concomitant reciprocally opposed change in P0.1 and PSV level.

Seven patients exhibited SCM muscle activity during which the mean P0.1 value was 4.1 (1.1) cm H2O. When no activity of these muscles was detected the P0.1 values were always lower than 2.9 cm H2O. During PSV Alberti et al. reported that P0.1 was closely correlated with the work of breathing and found, by extrapolation from linear regression analysis, a P0.1 value of 3.2 cm H2O corresponding to a work of breathing “threshold” level of 0.75 J/l of ventilation. In our study P0.1 was not modified in the absence of SCM muscle activity.

Our results corroborate this threshold in non-COPD patients. Berger et al. reported higher P0.1 values when patients were almost...
Changes in occlusion pressure and breathing pattern during PSV assistance. The authors thank Yves Goudard for his technical comments. The authors are grateful to Dr Laurent Brochard for very useful comments. The authors thank Yves Goudard for his technical assistance.

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