Determination of the Optimal Pressure Support Level Evaluated by Measuring Transdiaphragmatic Pressure

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The purpose of this study was to determine the optimum pressure support (PS) in six patients with respiratory failure. Esophageal pressure (Pe), gastric pressure (Pg), airway pressure, and transdiaphragmatic pressure (Pdi), obtained by subtracting Pe from Pg, were measured using a newly developed multiluminal nasogastric catheter. For each patient, different PS levels were selected every 20 minutes, and measurements were made at each PS level. We defined the optimum PS level as the level that showed the minimum Pe value. Respiratory rate (RR) decreased and tidal volume (VT) increased with an increase in PS level. RR and VT at the optimum PS were 17.7 ± 5.5 breaths per minute and 117.7 ± 4.5 ml/kg, respectively. Pdi decreased linearly with increasing PS level in all patients. Mean Pdi at the optimum PS was 4.2 ± 1.2 cm H2O. Based on the relationship between Pdi and PS level, we constructed an equation to estimate the optimum PS level as follows: Optimum PS level = ([Pdi during T-piece mode] – 4)/0.8. We conclude that Pdi measurement is helpful for titrating the required PS level.

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\[ \text{Paw} = \text{airway pressure}; \text{Pdi} = \text{transdiaphragmatic pressure}; \text{Pe} = \text{esophageal pressure}; \text{Pg} = \text{gastric pressure}; \text{Ppl} = \text{intrapleural pressure}; \text{PS} = \text{pressure support}; \text{PSV} = \text{pressure support ventilation}; \text{SIMV} = \text{synchronized intermittent mandatory ventilation} \]

Mechanical ventilation with pressure support (PS) can be characterized as a pressure assist during a spontaneous inspiration that decreases imposed work of breathing. Depending on the level of inspiratory pressure assist, ventilatory muscle effort is either partially or totally eliminated. However, an excess PS may be similar to total assisted mechanical ventilation with potential hyperinflation; an inadequate PS cannot reduce work of breathing and could result in respiratory muscle fatigue.

A simple way to determine the optimum PS is to use the pressure required to obtain the slowest regular respiratory rate (RR). The second method is to obtain a predetermined tidal volume (VT) used in a synchronized intermittent mandatory ventilation (SIMV) mode. Both methods were termed maximum pressure support ventilation (PSVmax) by MacIntyre. Another method is to find the PS level at which measured lung work and imposed work are least. Concerning the imposed work, the optimum PS is regarded as the level at which the total ventilatory system resistance to gas flow is nullified. However, in none of those methods can the changes in diaphragmatic activity with altering PS be evaluated. From the viewpoint of respiratory muscle fatigue, the optimum level of PS is, theoretically, the PS that maintains diaphragmatic activity without fatigue.

Diaphragmatic contractility cannot be assessed directly, and hence, the best indirect means is to obtain transdiaphragmatic pressure (Pdi) by subtracting intrapleural pressure (Ppl) from gastric pressure (Pg). The magnitude of Pdi could be a simple and useful marker of inspiratory effort for a given patient with severe respiratory disease undergoing pressure support ventilation (PSV).

The purpose of this study was to clarify the usefulness of Pdi measurement for titrating PS level by comparing Pdi with RR and VT at various PS levels.

Materials and Methods

Six patients requiring mechanical ventilatory support (7200a, Puritan-Bennett) were included in this study. Before conducting this study, informed consent was obtained from each patient following the guidelines set by the institutional human ethics committee. Clinical and respiratory data for each patient are shown in Table 1. Criteria for selection were as follows: (1) a spontaneous RR >8 breaths per minute when mechanical ventilatory support was discontinued; (2) stable chest roentgenogram and PaCO2 <50 mm Hg; (3) hemodynamic stability as evidenced by a systolic blood pressure >90 mm Hg and a regular cardiac rhythm. FiO2, and the SIMV rate were kept constant. Patients' spontaneous ventilatory rates exceeded the backup mandatory RR; patients were placed in a semirecumbent position, and no sedatives or narcotics were administered during the course of the study.

During mechanical ventilation, Pg and esophageal pressure (Pe) were simultaneously measured using transducers (DT-4812, Spectramed) attached by side arms to a newly developed multiluminal nasogastric tube (13 FG, custom made, Sumitomo Bakelite, Japan). The nasogastric tube has five lumina for sump, thermistor, suctioning, and monitoring Pg and Pe (Fig 1). The distal opening of the tube was placed in the stomach and the proximal opening was...
placed in the midthird of the esophagus. The position of openings for Pe and Pg was adjusted by chest and abdominal roentgenograms and confirmed further by each pressure tracing characteristic. The lumina for monitoring Pg and Pe were first flushed with physiologic sodium chloride solution and then kept patent with a slow and constant infusion of sodium chloride solution, 3 ml/h. Airway pressure (Paw) was measured at the distal end of the endotracheal tube. Pe, Pg, and Paw were continuously displayed on a bedside monitor (BSM 8500, Nihon kohden, Japan) and recorded on a multichannel thermal recorder (WS-900R, Nihon kohden, Japan). Pdi was calculated by subtracting Pe from Pg at the point of the negative peak in the Pe tracing.

The varying levels of PS were set for each patient. Each level of PS was used for a trial period of 20 minutes. Arterial blood gas tensions were analyzed (with an ABL 300, Radiometer, Copenhagen). Changes in PaO₂ and PaCO₂ (Δ PaO₂ and Δ PaCO₂) were calculated as the difference between the value of PaO₂ or PaCO₂ obtained at the end of the each test period and the value obtained during the lowest PS level. All recordings and measurements were obtained during the last two minutes of each trial. Measurements of Vt were made with a hot-wire flowmeter (Mini sensor, Minato, Japan) by integrating expiratory flows in the ventilatory circuit. Additionally, during a one-minute period of removal from mechanical ventilatory support on a T-piece mode in 100 percent oxygen, Pdi as the unassisted spontaneous ventilatory effort was measured in each patient.

Statistical analysis was performed using a linear regression technique to determine the relationship between ventilatory variables and levels of PSV. All values are expressed as mean ± SD.

### RESULTS

Changes in arterial blood gas tensions are shown in Figure 2. Altering PS level, Δ PaO₂, and Δ PaCO₂ varied from −14 to 36 mm Hg and from −3 to 5 mm Hg, respectively. We could not find a definite correlation between changes in arterial blood gases and

**Figure 1.** Scheme of the newly developed multiluminal nasogastric catheter.

**Figure 2.** Changes in PaO₂ and PaCO₂ compared with the value of PaO₂ or PaCO₂ during the lowest PS level.

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Table 1—Clinical and Respiratory Indices of Study Patients*

<table>
<thead>
<tr>
<th>Patient No. / Sex/ Age, yr</th>
<th>P(A-a)O₂, mm Hg</th>
<th>FIO₂, %</th>
<th>SIMV, min⁻¹</th>
<th>RR, min⁻¹</th>
<th>Diagnosis</th>
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<tbody>
<tr>
<td>1/F/21</td>
<td>571</td>
<td>70</td>
<td>10</td>
<td>22</td>
<td>HF, pneumonia</td>
</tr>
<tr>
<td>2/M/59</td>
<td>421</td>
<td>60</td>
<td>12</td>
<td>30</td>
<td>TAA, ARDS</td>
</tr>
<tr>
<td>3/M/64</td>
<td>274</td>
<td>40</td>
<td>3</td>
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<td>TAA, ARDS</td>
</tr>
<tr>
<td>4/F/60</td>
<td>605</td>
<td>100</td>
<td>0</td>
<td>30</td>
<td>TAA, ARDS</td>
</tr>
<tr>
<td>5/F/71</td>
<td>508</td>
<td>50</td>
<td>0</td>
<td>32</td>
<td>Pneumonia</td>
</tr>
<tr>
<td>6/M/67</td>
<td>498</td>
<td>50</td>
<td>0</td>
<td>30</td>
<td>Pneumonia</td>
</tr>
</tbody>
</table>

*HF = heart failure; TAA = thoracic aortic aneurysm; ARDS = adult respiratory distress syndrome; PH = pulmonary hemorrhage.
PSV levels.

Typical tracings of Pd, Pg, and Pd in eight situations at different levels of PS are shown in Figure 3 (patient 6). The magnitude of the negative deflection of Pd decreased with an increase in PS, resulting in a concomitant decrease in Pd. An excessively high level of PS caused a positive deflection of Pd. This phenomenon was observed in two patients (patients 3 and 6). A positive deflection of Pd suggests an excess PS at the end of inspiration. Thus, we defined the optimum PS level as the PS that showed the minimal negative or positive deflection of Pd during the late inspiratory phase; this was assumed to cause the least inspiratory effort.

The relationship between Pd and PS levels in six patients is shown in Figure 4. Pd decreased linearly with PS. The mean value of Pd at the optimum PS was 4.2±1.4 cm H2O and ranged from 2.7 to 6.8 cm H2O. Figure 5 shows the relationship between RR and PS. RR decreased linearly with increasing PS. RR at
the optimum PS ranged from 13 to 26 breaths per minute, and the mean value of RR at the optimum PS was 19.7±5.5 breaths per minute.

VT increased with increasing PS level as demonstrated in Figure 6. VT at the optimum PS varied widely from 6.4 to 18.8 ml/kg in each patient. The mean VT at the optimum PS was 11.7±4.5 ml/kg. The range of VT at PSV_{max} recommended by MacIntyre was marked in the hatched area in Figure 6. The values of VT at the optimum PS (Table 2) were different from VT at PSV_{max}.

We calculated the slope and probability of the relationships between respiratory parameters and PS levels (Table 2). The optimum PS and respiratory variables at the optimum PS for each patient are shown in Table 3.

### Table 3—Optimum PS and Respiratory Variables

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Optimum PS, cm H_2O</th>
<th>RR, min⁻¹</th>
<th>VT, ml/kg</th>
<th>Pdi, cm H_2O</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>18</td>
<td>14</td>
<td>12.6</td>
<td>3.4</td>
</tr>
<tr>
<td>2</td>
<td>15</td>
<td>23</td>
<td>7.4</td>
<td>2.7</td>
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<tr>
<td>3</td>
<td>15</td>
<td>13</td>
<td>6.4</td>
<td>4.1</td>
</tr>
<tr>
<td>4</td>
<td>25</td>
<td>24</td>
<td>12.2</td>
<td>4.1</td>
</tr>
<tr>
<td>5</td>
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<td>26</td>
<td>18.8</td>
<td>4.0</td>
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<td>6</td>
<td>20</td>
<td>18</td>
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<td>6.8</td>
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<tr>
<td>Mean</td>
<td>19.8</td>
<td>19.7</td>
<td>11.7</td>
<td>4.2</td>
</tr>
<tr>
<td>SD</td>
<td>4.8</td>
<td>5.5</td>
<td>4.5</td>
<td>1.4</td>
</tr>
</tbody>
</table>

### Discussion

PSV has been widely used for the purpose of assisting spontaneous breathing by decreasing inspiratory work of breathing. A problem with PSV is the need to individualize the PS requirement for each patient. RR, VT, the magnitude of inspiratory work, and total respiratory system resistance have been used as indices for optimizing PS. Our results showed that the optimum PS could be readily determined by measuring Pdi. MacIntyre and Leatherman defined the optimum PS as the ventilator setting at which RR was lowest or VT was 10 to 12 ml/kg. The VT at the optimum PS did not necessarily coincide with the values reported by MacIntyre and Leatherman since we titrated the PS level only to minimize Pdi reflecting diaphragmatic contraction. We suggest that the optimum PS could be fine-tuned by measuring Pdi as well as by gauging other respiratory parameters to reduce the patient's inspiratory effort.

Measurement of Pdi might be the most accurate method of assessing diaphragmatic function. Pdi is linearly related to the active diaphragmatic tension in dogs, and its waveform is reported to be related to electromyographic signs of diaphragmatic fatigue. Therefore, we chose Pdi as a marker of inspiratory effort generated by diaphragmatic shortening. In our study, before each pressure measurement was taken, gastric contents were suctioned to avoid a baseline shift of Pdi. Pdi measurement at the vital capacity was...
not measured since measurement of Pdi_max requires cooperation of the patient. Variability of Pdi_max resulting from inadequate cooperation shows the inherent operational difficulties in Pdi_max measurement under clinical settings. Therefore, we selected Pdi as a marker of inspiratory effort.

Pdi decreased linearly in accordance with the increase in PS. As shown in Figure 7, we express the relationship between Pdi and PS level by using the following equation (a):

\[ Y = AX + B, \]  

where A, B, X, and Y are slope, Pdi during T-piece mode, PS level, and Pdi, respectively. Thus, the equation (a) can be rewritten as follows:

\[ Y = -0.8 \times \text{(PS level)} + \text{Pdi during T-piece mode}, \]  

where the slope (-0.8) is taken from Table 2. Our result indicates that Y is assumed to be 4 when the optimum PS is applied (Table 3). Rearranging the equation (b), we get equation (c):

\[ P_{\text{di_optimum}} = (\text{Pdi}_{\text{spont}} - 4)/0.8. \]  

Therefore, if Pdi during T-piece mode is determined, the optimum PS could be determined by calculating equation (c). A further prospective study is needed for justification of equation (c) in clinical settings.

Caution must be paid when applying the equation (c) to the patient with severe respiratory muscle fatigue or paralysis. Pdi cannot be generated during diaphragmatic fatigue or paralysis. Therefore, the calculated optimal PS level may be too small compared with the required PS level. In such a case we recommend PSV_max when titrating PS level.

Vt and RR are also linearly correlated with PS as follows:

\[ P_{\text{di_optimum}} = (\text{RR}_{\text{spont}} - 19.7)/1.32, \]

where RR_{spont} is the spontaneous RR during T-piece mode.

\[ P_{\text{di_optimum}} = (11.7 - Vt_{\text{spont}})/0.24, \]  

where Vt_{spont} is Vt during T-piece mode. These equations would not necessarily eliminate the need for the measurements of Pe and Pg. Pdi is reduced by increasing Ps and converged to about 4 cm H2O in almost all patients (Fig 3). However, the values of SD in Vt and RR were greater than the SD in Pdi (Table 3). Thus, we regard the relation between Pdi and PS as more important than the relations between Vt or RR and PS.

PSV can reduce the patient's work of breathing to virtually zero. However, an excess PS shifts Pe upward and will not load the respiratory muscles sufficiently, resulting in atrophy of the respiratory muscles. An increased Vt due to high PS level might require longer expiration time to expel the inspired volume, resulting in a decrease in venous return. An excess PS could be judged by a positive deflection of Pe from the baseline level. Compression of the esophagus against the heart due to excess PS might have resulted in an increase of cardiac oscillations of Pe tracing as shown in Figure 3. Thus, positive deflection of Pe with increased cardiac oscillation also suggests excess PS.

Since Pdi is minimized when applying high PS level, inspiratory work is reduced and essentially the patient is being ventilated completely by the PS mode. This mode of ventilation is different from pressure-limited ventilation with regard to the initiation of the expiration. The expiration during PSV starts either at the point of a decrease of flow to 25 percent of the initial maximal flow or a decrease of demand flow to 5 L/min. Pressure-limited ventilation terminates the inspiration when Paw reaches the predetermined pressure value.

Patients who receive PSV can be weaned from the optimum PS by decreasing the inspiratory pressure assist to progressively lower levels as ventilatory failure resolves. Weaning is accomplished by gradually reducing the inspiratory pressure level, which allows the patient to acquire an increasingly greater proportion of his ventilatory work. The adequacy of weaning can be confirmed by adding conventional respiratory variables such as RR and arterial blood gas analysis. Weaning is complete when PSV level is just that required to overcome the resistance imposed by the tracheal cannula and demand valve. If the weaning process is too rapid compared with the recovering process of respiratory functions, tachypnea, abdominal paradox, sweating, and CO2 retention would appear, leading to respiratory muscle fatigue. Inappropriate weaning might cause a rapid increase in Pdi due to augmented inspiratory effort or a rapid decrease in Pdi, which means patient's intolerance with resultant respiratory muscle fatigue. Thus, Pdi measurement may have a significant influence on the course of weaning.
weaning from PSV.

Pdi measurement, originally, requires Ppl measurement. In practice a measurement of pressure inside the esophagus is a useful approximation to Ppl and can be obtained by having a patient swallow a catheter with a small balloon. We have developed a reliable and inexpensive multipurpose nasogastric catheter. The catheter has lumina for monitoring Pe and Pg in addition to the ordinary sump and avoids the introduction of an additional intragastric balloon. This method gives measurements of Pdi that correlate well with the esophagogastric balloon method. Using our new catheter, measurements of Pe and Pg were carried out with the minimum of patient discomfort.

In the semirecumbent position, however, the weight of the mediastinal contents will produce an artifact in the measurement of Pe. Therefore, Pe might be falsely high and Pdi might be falsely low. However, Pdi measured in the middle third of the esophagus, where we placed the opening hole of the Pe line, does not vary markedly with body posture. Furthermore, the downward force exerted by the mediastinum would influence all measured Pe evenly throughout this study without altering Pdi fluctuation and the slope between Pdi and PS level. Therefore, the topographic change of Pdi tracing was regarded as negligible.

In summary, Pdi measurement is helpful for titrating PS level. The optimum PS level can be determined by minimizing Pdi.

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