Respiratory Function during Pressure Support Ventilation*

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Pressure support ventilation (PSV) is a pressure assist form of mechanical ventilatory support that augments the patient's spontaneous inspiratory efforts with a clinician-selected level of positive airway pressure. To understand the effects of PSV on respiratory function, experiments were performed on 15 stable patients requiring synchronized intermittent mandatory ventilation (SIMV), as well as on a mechanical model simulating these patients' ventilatory systems. In the clinical study, gas exchange, airway pressures, blood pressure and heart rate were measured while SIMV was replaced by enough PSV to approximate the baseline SIMV tidal volume (Vt). Measurements were repeated while this PSV level was then reduced in three 5 cm H2O steps every 10 to 15 minutes. It was found that PSV was a reasonable form of mechanical ventilatory support in patients with spontaneous ventilatory drives. It improves patient comfort, reduces the patient's ventilatory work, and provides a more balanced pressure and volume change form of muscle work to the patient. The clinical significance of these properties during the weaning process remain to be determined.

The goals of mechanical ventilatory support are to supply adequate ventilation and oxygenation, reduce dyspnea, and promote rest and reconditioning of fatigued ventilatory muscles.1-7 Currently, the two most common modes for providing this support are assist-control ventilation (ACV) and synchronized intermittent mandatory ventilation (SIMV). Both ACV and SIMV are volume assist modes in that a patient inspiratory demand results in a clinician-controlled tidal volume and flow rate. ACV and SIMV differ, however, in that ACV provides a volume assist breath for every demand while SIMV intersperses assisted breaths with unassisted breaths. Both modes are effective in supplying alveolar ventilation to patients in acute respiratory failure. There are concerns, however, that these modes may not be optimal for patient comfort or muscle reconditioning during the weaning process from mechanical ventilatory support. These concerns are based on: 1) observations that the clinician-controlled volume and flows of the volume-assisted breath may not always be synchronous with the patient's ventilatory drive,8 and 2) theoretical considerations that the work characteristics of unassisted breathing during SIMV or during periods off ACV (ie, "t-tube trials") may not provide optimal ventilatory muscle reconditioning.7,9

Recently, the ability to augment spontaneous breaths with a variable amount of inspiratory positive pressure (ie, pressure assist) has been made available on several mechanical ventilators as "pressure support ventilation" (PSV, Fig 1). With PSV, a clinician-selected level of inspiratory airway pressure is provided as long as a patient demand exists. Thus, the patient has control of the ventilatory rate, the inspiratory assist time, and can interact with this pressure to determine the inspiratory flow and the delivered tidal volume. Pressure assist with PSV is designed to occur with every spontaneous breath although most ventilators also allow for additional volume-controlled or volume-assisted breaths. It should also be noted that although PSV is similar in concept to intermittent positive pressure breathing (IPPB), ventilators providing PSV offer larger inspiratory flows, servo control of the plateau of airway pressure, and more extensive ventilator alarm systems than older devices designed only for IPPB. The PSV is thus the preferable pressure assist mode for intubated patients.

PSV may have important effects on both patient comfort and muscle reconditioning. Specifically, a patient-controlled spontaneous breath assisted by PSV should interact better than clinician-controlled breaths with various intrapulmonary and thoracic cage reflexes thought to be important in producing the spontaneous ventilatory pattern and the sensation of dyspnea.10-20 PSV should also reduce the patient's work per breath21 and change the pressure and volume change relationships of that work (ie, work = ∫ pressure × volume change) to a lower pressure per volume change ratio. These changes in the characteristics of work might be particularly important in reconditioning an endurance muscle such as the diaphragm.7,9 Neither of these PSV effects, however, has been previously documented, and their implications in mechanical ventilatory sup-

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FIGURE 1. Schematic diagram of airway pressure in the distal endotracheal tube (top panel), air flow (middle panel), and lung volume changes (lower panel) in spontaneously breathing, intubated patients on a mechanical ventilator. The horizontal axis depicts time. The solid lines depict an unassisted breath while the dashed lines depict two levels of pressure assisted breaths with PSV. Note that under all conditions, an initial negative airway pressure change is required by the patient to open demand valves and initiate flow. Thereafter, continued negative airway pressure is required by the unassisted patient to maintain flow and deliver a tidal volume. With PSV, however, a plateau of pressure is produced that augments the patient’s inspiratory efforts to increase flow and tidal volume.

was designed to simulate the series arrangement of muscles and lungs in the ventilatory systems of these patients. This model was then used to describe the effects of increasing levels of PSV on the characteristics of ventilatory muscle work.

METHODS

Clinical studies

Fifteen stable patients requiring mechanical ventilatory support in our medical intensive care unit were selected for study. Criteria for selection were as follows: (a) a spontaneous ventilatory rate greater than 10 when removed from mechanical ventilatory support; (b) a stable or improving intrapulmonary disease process as evidenced by stable chest roentgenograms, an arterial PCO₂ less than 50 mm Hg and less than a 10 percent change in the alveolar-arterial oxygen difference P(A-a)O₂ over the previous 12 hours; and (c) hemodynamic stability as evidenced by a regular cardiac rhythm and a systolic blood pressure greater than 90 mm Hg without the use of intravenous pressors. All patients were receiving mechanical ventilatory support (Siemens 900c system) in the SIMV mode. Parameters had been set according to clinical guidelines in an effort to supply a VT of 10 to 15 ml/kg and a PaCO₂ of less than 50 mm Hg. All patients’ ventilatory rates exceeded the back-up mandatory rate.

During baseline mechanical ventilation, the following measurements were made: (a) mandatory and spontaneous ventilatory rates (f) and tidal volumes (VT) measured by integrating expiratory flows in the ventilator circuit; (b) mean, peak, and expiratory airway pressures (Paw) measured in the inspiratory circuit of the ventilator; (c) arterial hemoglobin saturation (SaO₂) determined from an infrared ear oximeter; (d) end tidal CO₂ (Pet CO₂) determined using an infrared sensor that sampled exhaled gases from the patient’s endotracheal tube; and (e) arterial blood pressure (BP) and pulse (HR) measured from an indwelling radial artery catheter using a standard strain gauge transducer and hemodynamic monitoring system. Additionally, during a two-minute period of removal from mechanical ventilatory support, determinations of the unassisted spontaneous ventilatory rate and tidal volume were performed. Following this, a determination of static respiratory system compliance (Crs) was made by dividing a mandatory VT by ΔPaw at the end of a three second inspiratory pause (ie, ΔPaw = inspiratory Paw – baseline expiratory Paw).

Inspiratory pressure assist with PSV was then added to the patient’s spontaneous ventilatory efforts using the same Siemens 900c system. Characteristics of this pressure assist were as follows: (a) -0.5 to -1 cm H₂O pressure in the inspiratory circuit triggered inspiratory flow to provide the set level of inspiratory pressure; (b) this pressure level was then maintained by a servo loop in the ventilator that continuously adjusted inspiratory flow; and (c) inspiratory pressure ceased when the inspiratory flow fell below 25 percent of peak flow. Expiratory pressure was not changed. The initial level of PSV was designed to result in a VT that approximated the patient’s SIMV delivered mandatory VT. All mandatory breaths were then stopped, and the patient was observed for 10 to 15 minutes for regularity of the spontaneous respiratory rate, changes in SaO₂, changes in the Pet CO₂, and subjective comfort. All baseline measurements were then repeated. The level of PSV was then reduced in three steps to 5, 10, and 15 cm H₂O pressure below the initial level. Baseline measurements were repeated 10 to 15 minutes after each change. Significant deterioration in SaO₂ (ie, >5%) or Pet CO₂ (ie, >5%) resulted in placing the patient back on the previous level of ventilatory support. For each patient, maximal PSV (PSVmax) was defined as that level which resulted in the slowest regular respiratory rate. Statistical analyses were performed using Student’s t test between paired measurements on SIMV and PSVmax, analysis of variance with Newman-Keuls test for multiple

port of patients are largely unknown.

Experiments were thus performed using PSV on patients, as well as on a mechanical model of the ventilatory system. In the clinical study, the effects of various levels of PSV on spontaneous ventilatory patterns, patient comfort, airway pressure, gas exchange, blood pressure, and heart rate were measured. In the mechanical model study, a two-compartment system
comparisons on SIMV and PSVmax, and linear regression techniques for ventilation parameters versus level of PSV. Significance was taken at p<0.05.

**Mechanical Ventilatory System Experiments**

A mechanical ventilatory system modelled upon the characteristics of our clinical study patients was used to study ventilatory muscle work requirements during unassisted and pressure assisted breaths with PSV. The specific model used was a two-compartment mechanical lung simulator with adjustable spring compliances. This device was modified to simulate the series arrangement of the ventilatory muscles and the lungs by linking the two compartments together with a rigid bar. One compartment then represented the muscles and one compartment represented the lungs. The muscle compartment was attached to a pressure limited, time cycled ventilator that supplied an adjustable inspiratory time, expiratory time, and accelerating pressure wave form. Pressure and flow in this compartment were measured by a small disc type pneumotachograph. The lung compartment was attached to the same Siemens 900c ventilator used in the clinical study. The model was set to simulate the impedances of intubated normal lungs, as well as those observed in the clinical study (see results below). The ventilatory pattern (ie, mean f and Vr) observed in the clinical study at each level of inspiratory pressure with PSV (see results below) was then simulated in the model by adjusting the ventilator parameters that drove the muscle compartment and the inspiratory pressure that supported the lung compartment. Under each ventilatory condition, two calculations were made: (1) muscle compartment work (W), was obtained by integrating the muscle compartment inspiratory pressure and flow for each breath over a one-minute period; (2) muscle compartment pressure and volume change work characteristics for each breath were expressed as the ratio of mean muscle compartment inspiratory pressure to tidal volume (Pn/Vt).

**Results**

**Clinical Results**

Characteristics of the 15 study patients along with the baseline mechanical ventilation parameters, arterial blood gases, respiratory system compliance, and ventilatory pattern during a brief period of unassisted ventilation are summarized in Table 1. All patients demonstrated tachypnea (ie, f of 19 or greater) during unassisted ventilation and most relied on mechanical ventilation for the majority of their ventilatory requirement.

PSVmax in these patients ranged from 13 to 41 cm H₂O. PSVmax was associated with a Vr equal to the SIMV Vr in nine patients while in the other six, a lower level of PSV was required to establish a regular breathing pattern. Ventilatory mechanics, gas exchange, blood pressure, and heart rate during SIMV and PSVmax are compared in Table 2. Minute ventilation (MV), oxygen saturation, end tidal CO₂, arterial blood pressure, and heart rate were similar during both forms of ventilation. Note, however, that PSVmax was associated with a significantly slower ventilatory rate, lower peak airway pressure, and higher mean airway pressure than SIMV. In eight of nine patients who could respond to the question, "Are you more comfortable with this form of ventilation (ie, PSVmax) or your previous form of ventilation (ie, SIMV)?", PSVmax was clearly more comfortable than a level of SIMV that provided a mean of 87 percent of the total MV (the ninth patient expressed no preference).

All patients tolerated the subsequent decreases in the level of PSV. The ventilatory effects of lowering the level of PSV are depicted in Figure 2. Linear regression analysis revealed significant correlation between level of PSV and MV (r = .44), Vr (r = .83), f (r = -.69),

<table>
<thead>
<tr>
<th>Sex/Age</th>
<th>Primary Diagnosis*</th>
<th>Minute Ventilation (ml/min/kg)</th>
<th>Arterial Blood Gas Values (mm Hg)</th>
<th>Unassisted Ventilation</th>
<th>Respiratory System Static Compliance (ml/cmH₂O)</th>
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<tbody>
<tr>
<td>M/19</td>
<td>Muscular dystrophy</td>
<td>Mandatory 150, Spontaneous 45</td>
<td>PaCO₂ 47, F(A-a)O₂ 10</td>
<td>Rate (BPM) 30, Tidal Volume (ml/kg) 3.2</td>
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<td>F/56</td>
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<td>35, 64</td>
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<td>M/74</td>
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<td>31, 150</td>
<td>25, 5.3</td>
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<tr>
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<tr>
<td>F/70</td>
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<td>39, 111</td>
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<tr>
<td>F/19</td>
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<tr>
<td>F/62</td>
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<td>50, 94</td>
<td>40, 1.9</td>
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</table>

*Diagnoses: COPD, chronic obstructive lung disease; AML, acute myelogenous leukemia; ARDS, adult respiratory distress syndrome; RMSF, rocky mountain spotted fever; CHF, congestive heart failure; BP fistula, bronchopleural fistula.†Alveolar-arterial oxygen difference.
Table 2—Ventilatory Parameters, Gas Exchange, and Hemodynamic Function During SIMV and PSVmax in 15 Patients (± SD)

<table>
<thead>
<tr>
<th></th>
<th>SIMV</th>
<th>PSVmax</th>
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<tr>
<td>Minute ventilation (ml/kg/min)</td>
<td>195.95 ± 18.71</td>
<td>193.52 ± 20.25</td>
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<tr>
<td>Vr (ml/kg)</td>
<td>12.9 ± 3.0 (mandatory)</td>
<td>11.8 ± 3.4*</td>
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<td></td>
<td>4.9 ± 2.9 (spontaneous)*</td>
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<tr>
<td>f (BPM)</td>
<td>23.5 ± 6.5†</td>
<td>16.4 ± 5.6*</td>
</tr>
<tr>
<td>Peak Paw (cm H2O)</td>
<td>33.5 ± 11.7*</td>
<td>29.4 ± 9.4*</td>
</tr>
<tr>
<td>Mean Paw (cm H2O)</td>
<td>7.3 ± 3.5*</td>
<td>9.6 ± 3.6*</td>
</tr>
<tr>
<td>SaO2 (%)</td>
<td>94.7 ± 2.7</td>
<td>94.8 ± 3.5</td>
</tr>
<tr>
<td>PetCO2 (mm Hg)</td>
<td>38.7 ± 6.5</td>
<td>36.1 ± 7.3</td>
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<tr>
<td>Systolic arterial pressure (mm Hg)</td>
<td>132.0 ± 30.1</td>
<td>122.4 ± 22.8</td>
</tr>
<tr>
<td>Heart rate (BPM)</td>
<td>106.3 ± 18.2</td>
<td>106.0 ± 19.5</td>
</tr>
</tbody>
</table>

*p<.05 between PSV value and SIMV value.
†Mean ± SD of mandatory and spontaneous f = 10.1 ± 3.3 and 13.4 ± 8.1, respectively.

and Paw (r = .67), but no significant correlation between level of PSV and SaO2, Pet CO2, HR or BP.

**Mechanical Ventilatory System Model Results**

The mechanical model was initially used to describe "normal" muscle work characteristics in our intubated patients by setting the lung compliance at 100 ml/cm H2O, the mean Paw at 5 cm H2O/L/s, and driving the muscle compartment to produce a Vr of 700 ml at ten breaths per minute (BPM). Assuming a dead space of 150 ml, this corresponded to an alveolar ventilation appropriate for our patient population of 5,500 ml/min.

The model was then adjusted to simulate observed compliances (ie, 15 and 45 ml/cm H2O) and the observed mean ventilatory pattern (ie, f and Vr for a typical 45 kg patient) over the range of mean PSV levels actually used (ie, 0 to 25 cm H2O). The muscle compartment was thus driven to produce the following ventilatory patterns: 327 ml x 31 BPM (PSV = 0 cm H2O), 346 ml x 28 BPM (PSV = 5 cm H2O), 370 ml x 25 BPM (PSV = 10 cm H2O), 400 ml x 22 BPM (PSV = 15 cm H2O), 439 ml x 19 BPM (PSV = 20 cm H2O), and 494 ml x 16 BPM (PSV = 25 cm H2O).

Figure 3 depicts these results. As can be seen, the normal system was characterized by a W of .62 kgm·min⁻¹, and a Pi/VT ratio of .012 cm H2O/ml. With decreasing compliance and tachypnea similar to that observed in the clinical study, unassisted muscle W and the Pi/VT ratio both increased substantially in the model. The addition of increasing levels of PSV, however, progressively decreased these muscle requirements for ventilation. Moreover, muscle W was reduced below normal at approximately 70 to 80 percent of PSVmax while the Pi/VT ratio approached normal near PSVmax.

**FIGURE 2.** Minute ventilation (MV, top panel), tidal volume (Vr/kg) and ventilatory rate (f, middle panel), and mean airway pressure (Paw, bottom panel) in 15 patients receiving varying levels of PSV as their only mechanical ventilatory support. The horizontal axis depicts applied PSV as a percentage of PSVmax (see text). Data are means ± SD of measured data. The open squares represent the mean values observed during SIMV in these patients. Numbers of patients in each group were as follows: 70 to 90 percent PSVmax = 15, 50 to 70 percent PSVmax = 9, 30 to 50 percent PSVmax = 8. Significant correlations were found between level of PSV and MV (r = .44), f (r = -.69), Vr (r = .83) and Paw (r = .67).

**DISCUSSION**

The results of this study demonstrated that, in these patients, inspiratory pressure assist with PSVmax resulted in mechanical ventilatory support comparable to SIMV but with a slower spontaneous ventilatory rate and more subjective comfort. Lower levels of inspiratory pressure resulted in less tidal volume augmentation and acceleration of the spontaneous ventilatory rate. A mechanical ventilatory system model patterned upon these patients' characteristics also demonstrated that PSV not only reduced ventilatory muscle work, but perhaps more importantly, altered the pressure/volume change characteristics of this work. The implications of these observations are discussed below.

**PSV Affects the Ventilatory Pattern and Patient Comfort**

For two reasons, a slowing of the spontaneous ventilatory rate and improved patient comfort should not be surprising when intermittent volume assisted
breaths are replaced by pressure assisted breaths in stable patients with intact ventilatory drives:

(1) The ventilatory pattern generator in the central nervous system is affected by arterial blood gas tensions and by neural pathways that sense the mechanical work of breathing (either directly or as a mean force requirement). The resulting ventilatory frequency and tidal volume pattern is thought to be that pattern which supplies the necessary alveolar ventilation for the minimum amount of muscle work. Thus, it should follow that as pressure support levels are progressively increased, the muscle work requirements per tidal volume should be progressively decreased, and the optimal ventilatory pattern should become progressively slower and deeper (Fig 2). In fact, since PaCO₂ (and thus alveolar ventilation) is comparable on PSVmax and SIMV, a PSVmax of approximately 16 BPM is predictable based on the PSVmax VT of 1.8 ml/kg and the observed minute ventilation requirements of 196 ml/kg/min on SIMV.

(2) Other mechanical factors affecting the ventilatory pattern and patient comfort include length-tension-time relationships in the chest wall and ventilatory muscles, as well as the activity of vagally mediated intrapulmonary receptors relating to stretch, irritation, or infiltration. Optimal comfort and “patient synchrony” with mechanical ventilatory support depend upon proper interaction of mechanical breaths with all of these factors. The pressure assist form of ventilatory support with PSV may provide better interaction with these factors than volume assist form of support. Specifically, a PSV breath allows the patient more control over inspiratory flow, inspiratory time, and tidal volume than volume assisted breaths. Moreover, since pressure assist with PSV is supplied with every spontaneous breath, muscle work, intrapulmonary stretch, and blood gas tensions should remain constant on a breath-to-breath basis. All of these factors probably contributed to the improvement in subjective comfort, and they offer a rationale for trying pressure assist modes in patients having difficulty “synchronizing” with mechanical ventilatory support.

**PSV Breaths Alter the Characteristics of the Patient Work of Breathing**

As with any form of mechanical ventilatory support, increasing levels of PSV should progressively decrease the patient’s work of breathing. Thus, it was not surprising that the mechanical ventilatory system model simulating these patients’ ventilatory system impedances indicated that ventilatory muscle work per minute was reduced to near normal at approximately 70 to 80 percent of PSVmax and could be reduced further at 100 percent of PSVmax (Fig 3). Perhaps of greater importance, however, was the demonstration in the mechanical lung model that increasing levels of PSV progressively changed the characteristics of the work (ie, work = ∫pressure × volume change) by reducing the ventilatory muscle pressure requirement and the pressure/volume change ratio of the work for each breath (Fig 3). Thus, mechanical ventilatory support with PSV not only appears to reduce the patients’ ventilatory muscle work, but it also appears to alter the characteristics of the work that remains.

These effects can be contrasted with those of SIMV at a similar level of absolute patient work by using the following calculations on the clinical and mechanical lung data: Taking the mean unassisted work per breath (ie, the work performed during each spontaneous breath on SIMV; Fig 3) and multiplying it by the mean spontaneous unassisted breathing frequency on SIMV (Table 2), one can calculate a mean workload to these...
patients of 0.624 kg·m·min⁻¹ during SIMV. The P/V ratio of this work is 0.44 cm H₂O/ml (Fig 3). On PSV, a mean absolute patient workload of 0.624 kg·m·min⁻¹ would occur at 12.1 cm H₂O of inspiratory pressure assist (Fig 3). The P/V characteristics of this work on PSV, however, is 0.025 cm H₂O/ml, only 57 percent of that observed during SIMV. The PSV can thus lower the pressure/volume change ratio of a given level of patient work during mechanical ventilatory support. This concept is depicted schematically in Figure 4.

How these effects of PSV might affect ventilatory muscle function in the clinical setting of respiratory failure is speculative. Clearly, ventilatory muscles fatigued by the increased impedances of lung disease need an initial period of rest along with appropriate nutritional and metabolic support.²⁸ During this time, most, if not all, ventilatory muscle work should be as close to zero as possible, and this is probably best accomplished by ACV or SIMV set near the patient's spontaneous rate. Following this initial rest, however, it is becoming increasingly apparent that it is important to initiate increasing levels of ventilatory muscle work so as to avoid disuse atrophy²⁹ and to begin muscle reconditioning.²⁴,²⁵,²⁶,²⁷,³⁰

The optimal timing and magnitude of this work are not well defined as muscle function is difficult to measure clinically. Thus, the amount of ventilatory work a patient is allowed to perform is generally determined by arbitrary criteria. Even less is known about the optimal pressure and volume change characteristics of this work. Indeed, these may have considerable importance if one considers that reconditioning can be aimed at different muscle properties. Specifically, ventilatory muscles, like other skeletal muscles, can either be strength conditioned (ie, develop more sarcomeres) by high pressure, low volume change work per breath or endurance conditioned (ie, develop an increase in mitochondrial density and more fatigue resistant fibers) by low pressure, high volume change work per breath.²⁴,²⁵,²⁶,²⁷,³⁰ Although both are probably important in ventilatory muscle reconditioning, ventilatory muscles are primarily an endurance system with a potential for high power output. Thus, the fixed ratio of high pressure, low volume change work during unassisted breaths with SIMV or with "t tube trials" (ie, stressing strength conditioning) may not be optimal. Rather a certain reduction in the pressure per volume change ratio of work with PSV (ie, stressing endurance conditioning) might offer theoretical advantages. Validation of this concept, however, will require difficult studies of ventilatory muscle function in the weaning phases of mechanical ventilatory support.

CONCLUSIONS

High levels of PSV (ie, inspiratory pressure assist levels providing Vr of 10 to 15 ml/kg) clearly can provide gas exchange comparable to volume assisted modes of ventilation (ie, SIMV) in stable patients with intact ventilatory drives. This high level of support is characterized by apparently improved patient comfort, slowed ventilatory rate, reduced patient work, and slightly higher mean airway pressures than SIMV. PSV, however, is an assist mode, and thus, it probably should not be used alone in patients with unstable ventilatory drives, lung mechanical function, or gas exchange function. Rather, the theoretical advantages of PSV appear to exist more during the weaning phases of mechanical ventilatory support when gas exchange is improving, re-establishment of patient control of ventilation is being encouraged, and ventilatory muscle reconditioning is being undertaken.³³ Under these circumstances, the better patient comfort/ventilator synchrony and more balanced work load of pressure assisted breaths may be preferable to intermittent

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**Figure 4.** Schematic diagram depicting how pressure assisting a patient's spontaneous breath can alter the pressure/volume change characteristics of that work. The horizontal axes represent pressure (patient generated on the pleural space to the left, machine generated on the ventilator circuitry to the right) and the vertical axes represent lung volume change for a single breath. The left panel depicts an unassisted breath as might occur under conditions of low lung compliance and high airway resistance. The hatched area under this curve represents patient workload. The right panel depicts a pressure supported breath in a lung with similar impedances and with a similar total workload to the patient. Patient and machine contributions to the total work are indicated by different shading. Note that for a similar level of patient total work, the patient's pressure/volume change work ratio for that breath is markedly reduced by pressure assisting the breath.

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volume assisted or controlled breaths. Further study, however, will be needed on the interactions of positive pressure ventilatory support and lung mechanical function and ventilatory muscle function before the true role of PSV is determined. Until then, weaning protocols utilizing primarily a gradual reduction in the level of pressure assisted breaths can only be considered an attractive theoretical alternative to conventional techniques.

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