Response of Ventilator-dependent Patients to Different Levels of Pressure Support and Proportional Assist

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The ventilator’s response to the patient’s effort is quite different in proportional assist ventilation (PAV) and pressure support ventilation (PSV). We wished to determine whether this results in different ventilatory and breathing pattern responses to alterations in level of support and, if so, whether there are any gas exchange consequences. Fourteen patients were studied. Average elastance (E) was 22.8 (range, 14–36) cm H₂O/L and average resistance (R) was 15.7 (range, 9–21) cm H₂O/L/s. The highest PSV support (PSVmax) was that associated with a tidal volume (VT) of 10 ml/kg (20.4 ± 3.2 cm H₂O), and the highest level of PAV assist (PAVmax) was 78 ± 7% of E and 76 ± 7% of R. Level of assist was decreased in steps to the lowest tolerable level (PSVmin, PAVmin). Minute ventilation, VT, ventilator rate (R), and arterial gas tensions were measured at each level. We also determined the patient’s respiratory rate (RR) by adding the number of ineffective efforts (ΔRR) to RR. There was no difference between PSVmin and PAVmin in any of the variables. At PAVmax, VT was significantly higher (0.90 ± 0.30 versus 0.51 ± 0.16 L) and RR was significantly lower (13.2 ± 3.9 versus 27.6 ± 10.5 min⁻¹) than at PAVmax. The increase in RR was largely related to a progressive increase in ineffective efforts on PSV as level increased (ΔRR 12.1 ± 10.1 vs 1.4 ± 2.1 with PAVmax); there was no significant difference in RR. The differences in breathing pattern had no consequence on arterial blood gas tensions. We conclude that substantial differences in breathing pattern may occur between PSV and PAV and that these are largely artifactual and related to different patient-ventilator interactions. Giannouli E, Webster K, Roberts D, Younes M. Response of ventilator-dependent patients to different levels of pressure support and proportional assist.

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Pressure support ventilation (PSV) (1, 2) and proportional assist ventilation (PAV) (3–5) differ fundamentally in the relation between timing and pressure amplitude of ventilator cycle on one hand, and timing and pressure amplitude of the patient’s inspiratory effort on the other hand. Because with PAV ventilator pressure output (airway pressure [Paw]) is proportional to instantaneous patient effort (Pmus), there is automatic synchrony between the ends of the patient’s effort and ventilator cycle. Furthermore, because Paw remains proportional to Pmus at all levels of assist, the total pressure applied (Paw and Pmus), and hence tidal volume (VT), remain under the control of the patient, even at the highest assist levels (3).

By contrast, with PSV there is no automatic linkage between the end of the patient’s effort and the end of the ventilator cycle. The timing of one relative to the other is governed in a complex way by many factors, the most important of these being the patient’s respiratory mechanics, the flow level used to cycle the ventilator off, and the magnitude of set PSV relative to Pmus (2, 6, 7). A part from the issue of synchrony, the fact that Paw is independent of Pmus renders VT more controllable by the ventilator, since at least one of the major VT determining forces (i.e., Paw) is independently controlled by the operator.

Because of these differences, it may be suspected that the response of VT and respiratory rate (RR) to changes in level of assist may differ in the two modes. In the present study we compare the response of ventilation, breathing pattern, and gas exchange to different levels of PAV and PSV in a group of ventilator-dependent patients with assorted pathology.

METHODS

Clinical Studies

We studied 14 patients who were placed on mechanical ventilation for a variety of disorders (Table 1). The study was done 2–7 d after intubation at a time when the clinical condition was stable (no need for vasopressors and no significant hemodynamic events or arrhythmias in the preceding 24 h). The protocol was approved by the Institutional Review Board and informed consent was obtained from patient or next of kin. All patients were ventilated on PSV prior to the study. The levels used and associated breathing pattern and blood gas tensions are

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shown in Table 1. All patients were ventilator-dependent as evidenced by development of respiratory distress upon attempts to reduce PSV. Apparatus. A heated pneumotachograph was inserted between the endotracheal tube and the Y connector to measure respired flow and volume. Paw was monitored at the Y connector. The exhalation valve was connected to a 10-L mixing chamber, and the Pco2 in the chamber (Paco2) was monitored with a capnograph (Normocap 200; DATEX, Helsinki, Finland). The flow, volume, Paw, and Paco2 were recorded on a computer disk.

In three patients (Patients 12–14) a fluid-filled esophageal catheter was inserted for direct monitoring of the patient’s efforts. The catheter was connected to a fluid-filled transducer (CDX press 3; COBE, Lakewood, CO). Saline was injected at a constant rate of 3 ml/h. The catheter was advanced so that the side hole was 35 cm from the nose. The catheter was then moved slightly up or down until there were strong cardiac oscillations and rhythmic negative respiratory efforts. The catheter was then moved slightly up or down to the level associated with minimal cardiac artifacts.

We used the Winnipeg ventilator. It has been described in detail earlier (4). Briefly, it is a piston-based ventilator that can provide PAV, PSV, and volume-cycled ventilation. Positive end-expiratory pressure (PEEP) is applied by attaching a PEEP valve to the exhalation valve and pressurizing the piston during expiration to a comparable level using an external control. In this fashion inspiratory flow begins as soon as Paw decreases below PEEP. The inspiratory cycle is flow-triggered, with the flow triggering level being externally adjustable. In the current experiments trigger sensitivity was set to the lowest level associated with minimal cardiac artifacts.

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In the PSV mode, the adjustable rate of rise of pressure (attack rate) was set to a time constant of 200 ms. The flow at which cycling off occurred was set to a fixed level of 0.1 L/s. In the PAV mode the levels of flow assist (FA) and volume assist (VA) were independently controlled using adjustable external analog dials.

Protocol. The Winnipeg ventilator was initially set in the PSV mode at identical fraction of inspired oxygen (Fio2), PEEP, and PSV levels to those used previously. A filter a few minutes to allow the patient to adjust to the new ventilator, the mode was switched to AC in the volume-cycled mode to measure respiratory elastance (E) and resistance (R). Vr was set to be the level observed on PSV or to 8 ml/kg, whichever was higher. Flow was set at 1.0 L/s. Back-up rate was increased until no triggering efforts were visible and the pressure waveform was reproducible (controlled mechanical ventilation [CMV]). Single-breath inspiratory hold maneuvers were carried out. A rough calculation of E and R was made at the bedside (R = ([peak pressure – plateau pressure]·V)/E = ([plateau pressure – PEEP]/Vr)). The relevant values were read off a needle meter on the face of the ventilator. More precise determinations of E and R were made at the end of the study from the computer stored data (see below).

Different levels of PSV and PAV were then studied. The order of testing (PSV first or PAV first) was randomized. The highest level of support was initially tested. For PSV, this was the level producing a Vr of approximately 10 ml/kg, which, according to MacIntyre and colleagues (8, 9), is associated with minimal work of breathing. For PAV the highest level was initially 80% of E and R, as determined in the CMV mode. A filter switching to PAV, end-inspiratory occlusions were repeated and E on PAV was recomputed (5). Subsequently the value of E obtained on PAV was used to set the high level of Vr (80% of E).* The high assist level was maintained for 10–15 min. With each mode, the level of assist was subsequently reduced in steps of 20% of the initial setting. Each step was maintained for 10–15 min. Decrements continued until a level was reached where some distress (accessory muscles use, chest wall indrawing, or paradox) became evident. At this point assistance was increased to the highest level and this was maintained for 20–30

#### TABLE 1

**PATIENT CHARACTERISTICS AND BASELINE DATA**

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<th>Patient No.</th>
<th>Age (y)</th>
<th>Weight (kg)</th>
<th>E (cm H2O/L · s)</th>
<th>Rr (cm H2O/kg · s)</th>
<th>Paco2 (mm Hg)</th>
<th>pH</th>
<th>ET</th>
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<th>PSV</th>
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Definition of abbreviations: AAA = abdominal aortic aneurysm; ARDS = adult respiratory distress syndrome; CABG = coronary artery bypass; CHF = congestive heart failure; CRF = chronic renal failure; CVA = cerebrovascular accident; E = elastance in cm H2O/L · s; ET = endotracheal tube size; MVA = motor vehicle accident; PEEP = positive end-expiratory pressure (cm H2O); PSV = pressure level during pressure support ventilation; RR = respiratory rate counted from ventilator cycles; Rr = total resistance in cm H2O/L/s; Vr = tidal volume.

* We used the value of E obtained during PAV (as opposed to that obtained during CMV), since it is the relevant elastance during breathing on PAV. Although on average E on PAV and on CMV are similar, there is some scatter, with some patients displaying higher E on CMV, and vice versa. In a systematic comparison in 48 patients with a range of elastance between 13 and 83 on CMV, the correlation (r) between E on PAV and on AC was 0.93, with a slope of 0.97 and intercept of 2.2 (not different from zero). The SD of the difference between the two measurements was 14% of the CMV value (for preliminary report, see Reference 27). Differences between E on PAV and on CMV in individual patients can be due to differences in Vr in the face of nonlinear elastic behavior, differences in T in the face of important viscoelastic behavior (28), or to differences in volume at the beginning of inspiration due to differences in degree of DH or to use of respiratory muscles during PAV (28).
Figure 1. Airway pressure (Paw), flow, esophageal pressure (Pes), and volume tracings from patient 12 at two levels of PSV (A and C) and two levels of PAV (B and D). (A) PSV = 18 cm H2O; (B) high PAV (actual % assist = 75%); (C) PSV = 12; (D) low PAV (actual % assist = 37%). (A) Upward pointing solid arrows represent transients in the flow and Paw tracings that were identified as ineffective efforts (see Footnote†). Downward pointing solid arrow indicates a flow transient that was questionable. Open arrow denotes an ineffective inspiratory effort (from Pes tracing) that was not identified from the flow and Paw tracing. Note that breathing patterns at the two PSV levels are substantially different (slow and deep in [A], rapid and shallow in [B]) despite similar patient’s respiratory rate (RRpat) (compare Pes tracing), and RRpat is similar in all four conditions despite different levels and types of support and different magnitude of inspiratory effort.

1 The criteria used to define ineffective efforts were based on the following considerations: To the extent that flow during the inspiratory phase is determined by (Paw – Palv)/R, a secondary increase in inspiratory flow during the inflation phase, despite the same or lower Paw, can only result if alveolar pressure (Palv) decreased or R transiently decreased. Changes in R due to changes in bronchomotor tone or to accumulation of thick secretions take place over a completely different time scale. Less viscid secretions can cause quick changes in R due to bubbling. The pattern, however, is readily recognizable. A decrease in Palv during an ongoing inflation, with progressively increasing volume (and hence, transpulmonary pressure), can only mean that pleural pressure (Ppl) decreased even more. It follows that a secondary increase in inspiratory flow, at similar or lower Paw, must be the result of a secondary reduction in Ppl. Likewise, transient departure of expiratory flow, in an inspiratory direction, from the flow trajectory established earlier during expiration can result either from a negative deviation in Palv, and hence Ppl, from its established trajectory or to an increase in R upstream or downstream from the Paw port. Changes in R upstream are subject to the same qualifications mentioned earlier. Changes in R of the expiratory tubing are excluded as the cause if Paw also decreases during the transient; an increase in external expiratory resistance would decrease flow but with an increase in Paw. It follows that a transient reduction in inspiratory flow along with reduction in Paw can only result from a transient reduction in Palv, and hence Ppl. The additional constraints used with both inspiratory and expiratory transients, of a minimum duration of 0.3 s and occurrence at fairly regular intervals consistent with respiratory rhythm, help eliminate other sources of nonrespiratory reductions in R (e.g., flow transients related to cardiac artifacts, movement, etc.). In addition, these constraints help exclude transients related to physical changes in bronchial or endotracheal tube secretions.

min. The procedure was then repeated for the other mode. The step during which distress occurred was not used for analysis.

Computer data were collected the last 3–5 min at each step. In 11 patients (Patients 1–11), an arterial blood sample was analyzed for PaCO2, PaO2, and pH at each step.

Analysis: The last 2 min of the computer-stored data were analyzed at each step to obtain average V˙I, ventilator respiratory rate (RRvent), inspiratory time of the ventilator cycle (T1) calculated from triggering to the point of resetting of pressure, respiratory duty cycle (T1/Ttot), V˙E, peak inspiratory pressure, and PEEP. CO2 production (V˙CO2) was computed from minute ventilation (V˙E) and mixed expired CO2 concentration. The computer data were also used to obtain the following:

1. Actual respiratory rate of the patient (RRpat). The Paw and ventilation (V˙) tracings at each step were examined for evidence of ineffective inspiratory efforts. At times during PSV the inflation cycle spanned more than one breath (Figure 1, top). The occurrence of a second inspiration during the same inflation cycle was defined as the occurrence of a secondary increase in inspiratory flow that is clearly distinguishable from signal noise (6, 7), lasting at least 0.3 s and associated with no increase (usually there is a small decrease) in Paw. Ineffective efforts occurring during the ventilator’s expiratory phase were counted when there was an unambiguous (i.e., clearly distinguishable from noise) transient reduction in expiratory flow associated with reduction in Paw and lasting at least 0.3 s (Figure 1, top) (6, 7). These dips were distinguished from artifacts by their duration and their occurrence at fairly regular intervals.
consistent with respiratory rhythm (Figure 1A) (see footnote on preceding page). The average number/min of these ineffective efforts was computed at each step (ΔR) and this was added to RRvent to arrive at true RR of the patient (RRpat). In the three patients in whom an esophageal catheter was inserted, RRpat was determined separately from the flow/Paw signals (as above) and from the esophageal pressure (Pes) signal. To avoid bias, the Pes signal was not displayed on the screen during determinations from flow/Paw, and vice versa.

2. Recalculation of respiratory mechanics. Bedside measurements of E and R were obtained from rapidly changing needle display and were not precise. The computer data were used to precisely calculate R and E on CMV and E on PA V. The values of E and R obtained in this fashion during CMV are reported in Table 1.

3. Calculation of FA and VA actually delivered during PA V. The external analog dials for FA and VA on the Winnipeg ventilator do not offer optimal resolution or accuracy. For these reasons we determined the actual assist delivered as opposed to relying on the dial position at the time of data collection. The actual FA and VA at each PA V step were computed by sampling Paw, V, and inspired volume at two points during randomly selected breaths. In these breaths one point was selected at peak flow while the other was selected near end inspiration, where inspired volume is high and flow is relatively low. The two sets of values were used to solve for the two unknowns (FA and VA) in the PA V assist equation: Paw – PEEP = FA · V + VA · V.

The values of FA and VA so obtained were expressed as percentage of R and E, respectively. For E we took the value of E-obtained PA V (see footnote on page 1717). For R, we took the value obtained during CMV, since there is no reliable way to non-invasively measure R on intermediate levels by interpolation. Comparisons between different PSV level patients were on prior to the study. The mechanical properties in this patient population (Rrs = 16 cm H2O/L/s and Ers in the operating range of 23 cm H2O/L) (Table 1). An average patient respiratory rate (RRpat) of 24 min-1 with a Ti/Ttot ratio of 0.4 was used. V, mechanical Ti, and relationship between RRpat and RRvent were computed at different levels of peak Pmus and assist.

**RESULTS**

Table 1 shows patient characteristics and baseline data on the PSV level patients were on prior to the study. The mechani...
cal abnormalities were mixed. All patients had abnormal gas exchange with a $P_{aO_2}/F_{iO_2}$ ratio of 116 to 295 (mean ± SD = 206 ± 48).

Figure 2A shows the relationship between the patient's respiratory rate (RRpat) determined from Pes and from the flow/Paw tracings in three patients in whom Pes was monitored. Most points fell very close to the line of identity. Figure 2B compares estimates of ineffective efforts ($\Delta RR$) using the two techniques. In most cases (virtually all PAV data and during the lowest one or two PSV levels) there were no ineffective efforts (< 1/min) with either technique. Large $\Delta RR$ values (up to 37/min) were obtained during higher PSV levels. Here, there was good agreement between the estimates of the two techniques ($r = 0.998$). There was a tendency for ineffective efforts to be slightly underestimated when flow/Paw was used (slope = 0.91). $\Delta RR$ determined from flow/Paw did not exceed $\Delta RR$ from Pes in any case.

Table 2 shows the results of some ventilatory and Paw data. The PSV level required to produce a Vt of approximately 10 ml/kg was 20.4 ± 3.2 cm H$_2$O. The actual Vt during PSV max was 10.2 ± 2.2 ml/kg. The lowest tolerated PSV level (PSV min) was 8.7 ± 3.0 cm H$_2$O. For PA V, the highest VA was 78 ± 7% of elastance and the highest FA was 76 ± 7% of resistance. At this level, $\Delta$PA was 16.2 ± 4.8 cm H$_2$O. At the lowest tolerated level delivered VA was calculated to be 31 ± 3.6% of E and FA was 55 ± 5% of R. Given the prevailing flow and Vt, as well as the values of R and E, we estimate that at PA V min the percent unloading was approximately halfway between the FA and VA delivered, or about 43% of the total work. At PA V min, $\Delta$PA was 11.1 ± 4.2 cm H$_2$O (Table 2).

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Figure 3. Average responses of tidal volume (Vt), ventilator rate (RRvent), patient's respiratory rate (RRpat), and number of ineffective efforts ($\Delta RR$) at different levels of PSV and PAV. Bars are SEM.

*Significantly different from comparable support level with the same mode. * Significantly different from value during minimum support with the same mode.

**Ventilatory* and Pressure Variables at Different Levels of PSV and PAV (n = 14)**

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</tr>
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<td>(4.1)</td>
<td>(3.5)</td>
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<td>(1.1)</td>
<td>(1.5)</td>
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<tr>
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<td>1.5</td>
<td>1.5</td>
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<td>(0.9)</td>
<td>(1.0)</td>
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</tr>
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<td>Ti/Ttot</td>
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<td></td>
<td>(0.06)</td>
<td>(0.07)</td>
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</table>

All values represent mean (± SD).

$\Delta$Paw = differences between peak airway pressure during inspiration and positive end-expiratory pressure (PEEP). Ti, Te, and Ti/Ttot refer to ventilator's inspiratory and expiratory times and the inspiratory duty cycle. All statistical comparisons are results of two-way ANOVA for repeated measures with two-way interaction.

* Other ventilatory variables are shown in Figure 3.
† Significantly different from level 1 in the same mode.
‡ Significantly different from same level of PAV.
RRvent decreased below 20/min, but the range of RRpat was still quite large (12 to 46/min). With PAV, with one exception where ΔRR was 7.0/min, the data points remained very close to the line of identity.

Ventilator inspiratory time (T_I) increased progressively with level of PSV, with T_I exceeding 2.0 s in seven patients. T_I increased only slightly with PAV level. The difference in T_I response between PSV and PAV was significant at the higher two levels (Table 2). T_E showed corresponding changes, increasing markedly with PSV but not with PAV. There was no change in T_I/T_tot as a function of mode or support level.

Minute ventilation did not show consistent changes with level of assist or with mode (Table 3).

Changes in PaO₂, PaCO₂ and pH with assist level in the two modes were comparable (Table 3). V̇D/VT decreased with level of assist in both modes. The decrease was more pronounced in PSV. V̇CO₂ did not change with mode or level of support (Table 3).

Computer Simulation Results

In both panels of Figure 5, the diagonal line marked zero represents the relationship between peak Pmus and V̇T in the absence of assist. With PAV, as percent of assist increased, the slope of the relation increased. In all cases a stable solution was reached with RRpat:RRvent ratio of 1:1 and the ventilator cycle terminating within 0.1 s of the end of inspiratory effort.

In the PSV mode the relation was variable and was affected by PSV level and intensity of effort. At PSV 5, the assist simply caused a parallel upward shift and the RRpat:RRvent ratio remained 1:1. At higher levels the response displayed three

**TABLE 3**

GAS EXCHANGE VARIABLES AT DIFFERENT LEVELS OF PSV AND PAV (n = 11)

<table>
<thead>
<tr>
<th>Mode</th>
<th>Min 1</th>
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<th>3</th>
<th>Max 4</th>
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<tr>
<td>V̇E, L/min</td>
<td>PSV</td>
<td>12.2 (4.5)</td>
<td>11.4 (4.0)</td>
<td>10.7 (3.1)</td>
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<td>12.6 (4.5)</td>
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<tr>
<td>PaO₂, mm Hg</td>
<td>PSV</td>
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<td>99 (18)</td>
<td>100 (22)</td>
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<td>96 (19)</td>
<td>99 (18)</td>
<td>100 (20)</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>PSV</td>
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<td>41.6 (9.7)</td>
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<td>40.7 (8.0)</td>
<td>41.8 (8.1)</td>
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<tr>
<td>pH</td>
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<td>7.41 (0.07)</td>
<td>7.41 (0.07)</td>
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<tr>
<td>V̇CO₂, ml/min</td>
<td>PSV</td>
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<td>289 (79)</td>
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<td>290 (63)</td>
<td>295 (66)</td>
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<tr>
<td>V̇O₂/VT</td>
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<td>0.49 (0.07)</td>
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<td>PAV</td>
<td>0.58 (0.07)</td>
<td>0.55 (0.07)</td>
<td>0.56 (0.07)</td>
</tr>
</tbody>
</table>

All values represent mean (± SD). V̇CO₂ = rate of CO₂ production.

* Significantly different from level 1 in the same mode.

† Significantly different from same level in PAV, two-way ANOVA for repeated measures with two-way interaction.
phases. A high peak Pmus there was a vertical shift and a
continued R Rpat:R R vent ratio of 1:1. A peak Pmus decreased
to a critical level, which was higher at higher PSV lev-
els, a transitional zone developed where the relation between
Pmus and VT was biphasic; VT decreased sharply as Pmus de-
creased, followed by a rapid increase as Pmus decreased fur-
ther. A t the beginning of this transitional zone, ventilator
cycle began extending progressively more into neural expira-
tion, reducing the time available for emptying and resulting
in progressively more dynamic hyperinflation (DH). Initially,
efforts continued to be able to trigger the Paw cycle, and
R R pat:R R vent ratio remained 1:1. However, VT decreased
sharply as DH increased. A t slightly lower levels of peak Pmus
in this transitional phase a steady state was not reached; with
successive neural cycles, ventilator cycle extended progressively more into neural T E and end-expiratory volume pro-
gressively increased. A fter a number of cycles, one effort fails
to trigger PSV, allowing volume to return to passive FRC. The
sequence then repeats (notice 23:22 ratio at the trough in the
PSV 20 line). A s Pmus decreased further, within this transi-
tional zone, mechanical T i increased further and a larger frac-
tion of breaths failed to trigger PSV. In this region average VT
increased (as Pmus decreased), in part because more breaths
started near passive FRC (less DH) as a result of preceding
nontriggered efforts and in part because of the longer mechan-
cal T i. Beyond this transitional zone, VT reached a constant
level that was independent of Pmus with a stable 2:1 rhythm.
Here, every triggered breath begins near passive FRC and me-
canical T i is long enough to permit equilibration between ap-
plied pressure and elastic recoil. VT equals PSV/E Rs.

Whereas Figure 5 shows the result for the case where the
patient’s R R and mechanics are similar to the average patient
in this study, other simulations (not shown) using different pa-
tient characteristics show that a higher R R pat and a longer
time constant (R s/E s) cause the transitional zone to occur at
relatively higher Pmus and the R R pat:R R vent ratio at mini-
mal Pmus to be greater (e.g., 3:1, 4:1, etc.), and vice versa.

DISCUSSION

The present study shows that the breathing pattern responses
to different levels of PSV and PAV are substantially different
and that these differences are largely artifactual, reflecting the
manner in which the ventilator responds to changes in patient
effort in the two modes.

Technical Considerations

Use of flow to estimate ineffective efforts. Our use of flow and
Paw to identify ineffective efforts requires justification. The
criteria used were based on straightforward physical principles
and practical consideration of the features of other possible
causes of such flow transients. We therefore feel that with the
criteria used here there is little possibility that ineffective ef-
fiants will be wrongly identified. This was borne out by the re-
sults in the three patients in whom efforts were also directly
identified from Pes; in no case did the number of ineffective
efforts identified from flow exceed the number identified from
Pes. Difficulties may arise where the magnitude of the flow
transient is very small or its duration is borderline (e.g., tran-
sient marked by downward pointing arrow, Figure 1A). These
features, however, reflect feeble and/or very brief negative
transients in intrapleural pressure (Ppl) (see corresponding
Pes tracing, Figure 1A). Such transients would be question-
able regardless of whether one uses flow or Ppl.

Whereas overestimation of ineffective efforts using the cur-
rent criteria would be quite unlikely, underestimation is possi-
ble under two circumstances. First, if an inspiratory effort occurs at the very beginning of the ventilator's expiratory phase, before a clear expiratory flow trajectory has been established, it may simply truncate the peak of the expiratory flow and not be clearly recognized as an independent flow transient (e.g. effort marked by open arrow, Figure 1A). Second, where expiratory flow limitation exists and Ppl is in excess of the value required to generate maximum flow, reduction in Ppl may not result in reduction in expiratory flow. The three patients in whom ineffective efforts were identified independently by the two techniques represent extreme examples of dysynchrony with PSV. The results in these patients (Figure 2B) suggest that underestimation of ineffective efforts by the flow method is acceptably small.

Comparability of level of assist with PSV and PAV. The choice of the lowest level of assist was based on a clinical judgment by the experimenters (E.G. and K.W.) that the patient could not sustain a further reduction in support. This is admittedly subjective. However, since no difference was identified in any variable at minimum assist, the issue of whether the assist level was comparable is moot. The situation is, however, different at the higher levels of assist where substantial differences in breathing pattern and ∆RR were observed. It may be argued that the observed differences in breathing pattern and ∆RR were related to a relatively lower level of assist with PAV. We believe this to be highly unlikely for the following reasons:

1. Arterial PO2, PCO2, and pH were virtually identical at level 4 (Table 3). To the extent that these are important (if not the most important) determinants of inspiratory effort, there is no reason to believe that patients had a higher respiratory drive with PAV.

2. Although the operational target for PAVmax was 80% of E and R, the actual assist delivered, determined posthoc, ranged from 65 to 95%. There was no evidence that increasing PAV in the range from 80 to 95% resulted in a different response to PAV. In fact, in three patients in whom percent of assist exceeded 90%, the differences in Vr between PAVmax and PAVmin were zero, 0.06, and 0.05 L, not any greater than the average increase in Vr (0.06) (Figure 3) and in each of these patients ∆RR was zero. This suggests that using a higher PAVmax would not have altered the results.

3. Although ∆Paw at PAV max was only 80% of ∆Paw at PSV max (16.2 ± 4.8 versus 20.4 ± 3.2 cm H2O) (Table 2), the pressure assist with PAV was delivered more than twice as often (Rvent = 27.6 ± 10.5 with PAVmax versus 13.2 ± 3.9 with PSV max) (Figure 3). The product ∆Paw Rvent at PAV max was 167% of the corresponding value at PSV max.

4. Breathing pattern at PSV 3 remained different from PA V max where ∆Paw was similar (16.2 ± 4.8 versus 16.4 ± 2.7 cm H2O) (Table 2). Thus, at PSV 3, Vr was larger (p < 0.02), Rvent was lower (p < 0.005) and ∆RR was greater (p < 0.005) than at PAV max.

Responses to Different Levels of PSV and PAV

The results observed during PAV are very similar to those reported previously (15); the changes in Vr and respiratory rate are quite small over a wide range of assist in the tolerable range. The response to different levels of PSV in the current study is also similar to earlier findings (1, 16–19; Vr progressively increases and ventilator rate progressively decreases as PSV level is increased. The advantage of the present study is that both modes were used in the same patients, thereby allowing us to determine whether the different responses are artefactual or represent differences in respiratory control with the two modes.

Differences in respiratory rate. The responses of ventilator rate to different levels of PSV and PAV were substantially different; the decrease in Rvent with PSV was nearly four times greater than with PAV (29.6 to 13.7 with PSV versus 31.9 to 27.6 with PAV) (Figure 3). These differences, however, were largely artefactual; the responses of RRpat were substantially smaller in absolute terms and not significantly different between the two modes (Figure 3). The decrease in Rvent with PSV was predominantly related to a progressively greater number of ineffective efforts (∆RR) (Figure 3).

That discrepancies between Rvent and RRpat are likely to occur with PSV was theoretically predicted earlier (6, 7) and was well demonstrated in practice in several recent studies (20–22). It was also predicted that PAV would be less likely to result in such discrepancies (6, 7). The present study confirms this prediction. As shown by computer simulation of the two modes (6, 7, current study), the main reason for this to occur with PSV is the tendency for ventilator Ti to outlast neural Ti, particularly when the mechanical time constant (R/E) is long. This results in reduction in the time available for expiration, and hence inadequate emptying prior to the onset of the next effort. Computer simulations have also shown that the extent to which ventilator Ti exceeds neural Ti, and hence the frequency of unsuccessful efforts, should increase as the level of PSV increases (6, 7). This prediction is also borne out by the present results: ∆RR progressively increased with PSV level.

In a previous study using different levels of PA V, we observed that RRpat covered a wide range (18 to 33 min⁻¹) despite high levels of assist (15). The present results confirm these previous findings and extend them in two respects. First, the same conclusion applies during PSV if one considers the patient's rate as opposed to Rvent. Second, the range of RRpat observed in the current study is considerably larger. Thus, at the highest assist level, RRpat ranged from 12 to 46 min⁻¹ with PSV, and 8 to 46 min⁻¹ with PAV (open circles, Figure 4). More importantly, in several patients with marked tachypnea at low levels of support, RRpat changed little over the entire range of assist (Figure 4). This suggests that in some patients tachypnea may be largely due to abnormal tachypneic influences unrelated to respiratory load.

Differences in tidal volume response. Although Vr increased with level of assist in both modes, the increase was substantially more in the case of PSV (Figure 3). The large increase in Vr with PSV cannot be attributed to unusually high levels of PSV; even at PSV max, PSV level was well within the range used in practice both in terms of absolute pressure (20.4 ± 3.6 cm H2O) (Table 2) and in terms of associated Vr (10.2 ± 2.2 ml/kg). Likewise, the small increase in Vr with PAV was not due to low levels of assist (see Technical Considerations).

We believe that the difference in Vr response with the two modes is largely related to different patient-ventilator interactions. While with both modes inspiratory effort decreases progressively as assist level increases (16–19, 23–25), the response of the ventilator to this downregulation of Pmus is very different in the two modes. The results of computer simulation carried out here (Figure 5), where patient characteristics were matched to those of the average patient in this study and ventilator characteristics were similar, provide a framework for interpreting the experimental results. For simplicity, only the changes between minimum and maximum assist will be discussed. At PA V min average Vr was 0.45 L at an average assist level of approximately 45%. This is represented by the solid circle on the 45% line (Figure 5, right panel). Increasing the
assist to an average of 75% (actual increase was to 77%) would, in the absence of downregulation of $P_{\text{mus}}$, immediately result in doubling of $V_T$ with continued 1:1 synchrony (open square, Figure 5, right panel). $P_{\text{aCO}_2}$ would rapidly decrease as a result of the increased $V_E$ and decreased $V_D/V_T$ (due to larger $V_T$). Peak $P_{\text{mus}}$ decreases as a result, causing $V_T$ to decrease along the 75% line. A new steady state is reached (open circle) at a point where the decrease in $P_{\text{aCO}_2}$ is commensurate with the decrease in $P_{\text{mus}}$ (as governed by $CO_2$ responsiveness) and the change in $V_E$ is commensurate with the change in $P_{\text{aCO}_2}$. It can be seen from this analysis that a substantial increase in $V_T$ with $PAV$ is possible only if RR decreases substantially, thereby minimizing the decrease in $PCO_2$, or $P_{\text{mus}}$ fails to downregulate as $PCO_2$ decreases (e.g., in the presence of important nonchemical sources of drive).

In our patients, $V_o/V_T$ decreased slightly, while metabolic rate did not change as a result of increasing $PAV$ support from minimum to maximum (Table 3). As a result of the small decrease in $V_o/V_T$, $P_{\text{aCO}_2}$ decreased by 2.8 mm Hg, despite a small reduction in $V_E$ (13.4 to 12.6 L/min) (Table 3). A tidal volume $V_T$ actually increased from 5.6 to 5.9 L/min, which accounts for the decrease in $P_{\text{aCO}_2}$. RR$_{\text{pat}}$ decreased from 30 to 26.4 min$^{-1}$, likely the result of the decrease in mechanical load and/or the small decrease in $P_{\text{aCO}_2}$. Had RR$_{\text{pat}}$ decreased less, $V_T$ would have increased even less than it did. The opposite, of course, would occur if RR$_{\text{pat}}$ decreased more. The decrease in estimated $P_{\text{mus}}$ between $PAV_{\text{min}}$ and $PAV_{\text{max}}$ (14.0 cm H$_2$O to 8.2 cm H$_2$O, Figure 5, right panel) is likely related to the decrease in $P_{\text{aCO}_2}$, although a reduction in other sources of respiratory drive cannot be ruled out.

The solid circle in the left panel of Figure 5 represents the case at PSV$\text{min}$ (average $V_T = 0.44$ L at PSV of approximately 10 cm H$_2$O) (Table 2). Increasing PSV from 10 to 20 cm H$_2$O would have, in the absence of $P_{\text{mus}}$ downregulation, resulted in an increase in $V_T$ to 0.73 L with maintained 1:1 synchrony (open square, Figure 5, left panel). $P_{\text{aCO}_2}$ would decrease sharply and $P_{\text{mus}}$ would decrease accordingly. As a result, $V_T$ would initially decrease sharply. However, if $V_T$ at the trough of the transitional phase is not commensurate with a steady state, $P_{\text{mus}}$ continues to decrease. $V_T$ rises again, along with development of increasing degrees of nonsynchrony and prolongation of mechanical $T_I$. The final steady state would again be reached when the decrease in $P_{\text{aCO}_2}$ is commensurate with the decrease in $P_{\text{mus}}$ and the increase in alveolar volume is commensurate with the decrease in $P_{\text{aCO}_2}$. In the average patient represented in Figure 5, the trough of the $V_T$ response at PSV 20 would not be consistent with a steady state. $V_T$ at this point was 0.58, 26% greater than $V_T$ at PSV$\text{min}$. Even ignoring the lower $V_o/V_T$, this would have resulted in a 10 mm Hg reduction in $P_{\text{aCO}_2}$. $P_{\text{mus}}$ could not stabilize at the trough level and must decrease further, forcing the nonsynchrony and the secondary increase in $V_T$. The equilibrium point thus occurs at a much higher $V_T$. On average, in our patients at this equilibrium point (PSV$\text{max}$), approximately one-half of inspiratory efforts failed to trigger ($\Delta RR = 12.1$, RR$_{\text{pat}} = 25.3$ min$^{-1}$) (Figure 3). It is of interest to note that according to the simulation presented here, mechanical $T_I$ would increase from 0.9 s to 1.9 s between PSV$\text{min}$ and PSV$\text{max}$, despite a constant neural $T_I$ in the simulation. This is quite similar to what was actually observed (0.9 to 2.1 s) (Table 2).

The simulation data shown in Figure 5, left panel, also provide an explanation for the occasional occurrence of rapid dramatic changes in ventilator output during PSV. Figure 6 provides an example. In this case, breathing pattern, as judged by

![Figure 6](image-url)
ventilator output, suddenly and spontaneously changed from slow, deep breathing to rapid, shallow breathing. The increase in ventilator rate was substantially greater than the increase in RR - pat (arrows), reflecting better synchrony. The improved synchrony was, in turn, likely related to an increase in patient effort. Note that with improved synchrony, \( V_T \) decreased. The transitional zone demonstrated by the computer simulation implies that small changes in \( P_{\text{mus}} \) may cause large changes in ventilator \( V_T \) and RR vent when they occur in a critical \( P_{\text{mus}} \) range.

The above analysis indicates that the differences in the experimental results can be readily explained by the different patient-ventilator interactions in the two modes. It is to be noted that the expected increase in \( V_T \) upon transition from \( \text{PAV}_{\text{min}} \) to \( \text{PAV}_{\text{max}} \) in the absence of \( P_{\text{mus}} \) downregulation (difference between solid circle and open square, Figure 5, right panel) is substantially larger than the corresponding expected increase upon transition from \( \text{PSV}_{\text{min}} \) to \( \text{PSV}_{\text{max}} \) (0.43 L in right panel versus 0.26 L in the left panel, Figure 5). This further supports our contention that the smaller \( V_T \) response with \( \text{PAV} \) was not due to a relatively smaller range of assist.

**Clinical Implications**

1. The present study, along with earlier theoretical (6, 7) and experimental (20–22) findings, indicates that RR vent should not be used as a surrogate for RR pat in the PSV mode, whereas RR vent is a reliable surrogate of RR pat during \( \text{PAV} \) support.
2. A high RR pat, as high as 46/min, on either \( \text{PAV} \) or \( \text{PSV} \) need not reflect inadequate support. In some patients respiratory rate is fairly insensitive to level of support. This conclusion is further supported by recent findings that respiratory rate is not a good predictor of work of breathing or pressure-time product during PSV (16, 26).
3. During PSV, particularly in patients with high resistance, breathing pattern, as judged by ventilator output, can undergo dramatic changes in response to minor changes in the patient's respiratory motor output or \( \text{PSV} \) level. These changes may be artifactual and should not be interpreted as a major change in the patient's inherent pattern of breathing or level of distress without independent confirmation.
4. A high synchrony between patient and ventilator is much better preserved with \( \text{PAV} \), the clinical significance of this feature is not clear. There is very little impact on \( P_{\text{O}_{2}} \) and it does not appear that synchrony, or lack thereof, materially affects gas exchange as judged by the \( P_{\text{A}_{2}}/F_{\text{I}_{2}} \) ratio.

In summary, the breathing pattern response to changes in assist level with \( \text{PSV} \) and \( \text{PAV} \) are substantially different. These differences are, however, largely artifactual and have little impact on gas exchange. The present study also confirms that breathing pattern at high levels of assist is highly variable with some patients displaying respiratory rates in the forties with very small \( V_T \) (approximately 0.3 L). Such patterns in the course of weaning need not signify excessive work.

**References**