Ventilator Waveforms and the Physiology of Pressure Support Ventilation

Dean R Hess PhD RRT FAARC

Pressure support ventilation (PSV) is a commonly used mode. It is patient-triggered, pressure-limited, and (normally) flow-cycled. Triggering difficulty occurring during PSV is usually due to intrinsic positive end-expiratory pressure. The airway pressure generated at the initiation of inhalation is determined by the pressure support setting and the pressure rise time (pressurization rate) settings on the ventilator. The rise-time setting is clinician-adjustable on many current-generation ventilators. Flow delivery during PSV is determined by the pressure support setting, the pressure generated by the respiratory muscles, and respiratory system mechanics. The delivered tidal volume is determined by the area under the flow-time curve. Patient-ventilator dyssynchrony may occur during PSV if the flow at which the ventilator cycles to exhalation does not coincide with the termination of neural inspiration. The newer generation ventilators offer clinician-adjustable flow-termination during PSV. Ventilator waveforms may be useful to appropriately adjust the ventilator during PSV. *Key words: acute respiratory failure, mechanical ventilation, pressure support ventilation.*
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Introduction

Pressure support ventilation (PSV) is patient-triggered and pressure-limited. It is primarily flow-cycled, meaning that the ventilator normally cycles to exhalation when the flow decreases to a ventilator-determined level. PSV is a commonly used ventilation mode. One survey reported that PSV was used as a weaning mode with 45% of patients in the United States.[1] In an international survey of mechanically ventilated patients, PSV was used with 21% of patients during weaning from mechanical ventilation.[2] PSV is generally considered a simple ventilation mode. In this paper I will use ventilator waveforms to illustrate the technical and clinical characteristics of PSV.

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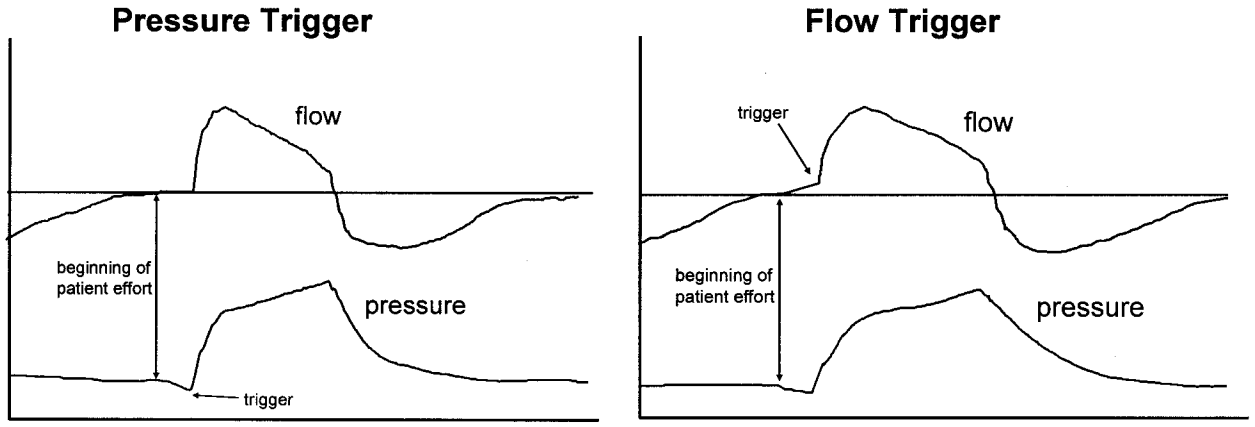

Fig. 1. Pressure trigger (left) and flow trigger (right). Note that with pressure-triggering the pressure-supported breath is triggered when the pressure drops below baseline. With flow-triggering the pressure-supported breath is triggered when the flow rises above baseline, indicating an inspiratory effort from the patient.

The Ventilator Trigger

PSV is patient-triggered. If the patient becomes apneic, a modern ventilator will detect the apnea, initiate backup ventilation, and alarm to alert the clinician. However, the use of PSV assumes that the patient is capable of initiating an inspiratory effort. Usually the patient's effort is detected by a pressure trigger or a flow trigger.

Pressure-triggering requires patient effort sufficient to decrease airway pressure from the end-expiratory level to a threshold setting (pressure sensitivity) on the ventilator (Fig. 1). Pressure sensitivity settings from −0.5 cm H_2O to −2.0 cm H_2O are safe and effective with most patients. With flow-triggering, breath initiation is based on a flow change in the ventilator circuit beyond some predetermined threshold (flow sensitivity) (see Fig. 1). Flow sensitivity settings of 1–5 L/min are typical. Sassoon et al have described triggering in detail.[3–6] During the pre-trigger phase, the patient generates effort prior to ventilator response. This delay may produce patient-ventilator dyssynchrony if prolonged. The trigger sensitivity settings and the responsiveness of the ventilator to the trigger affect the pre-trigger phase. The post-trigger phase, however, is affected by other ventilator settings (rise time and the pressure support setting).

A number of studies have compared pressure triggering and flow triggering.[7] With older generations of ventilators a common finding was that flow-triggering was superior to pressure-triggering. However, Tutuncu et al,[8] using the Siemens Servo 300 ventilator, and Goulet et al,[9] using the Puritan-Bennett 7200 ventilator, reported similar patient responses with flow and pressure-triggering during PSV. Aslanian et al[10] reported a modest benefit from flow-triggering with PSV, but suggested that the benefit may be too small to affect clinical outcomes (Fig. 2). Moreover, they reported that differences between pressure triggering and flow triggering were related primarily to the post-trigger

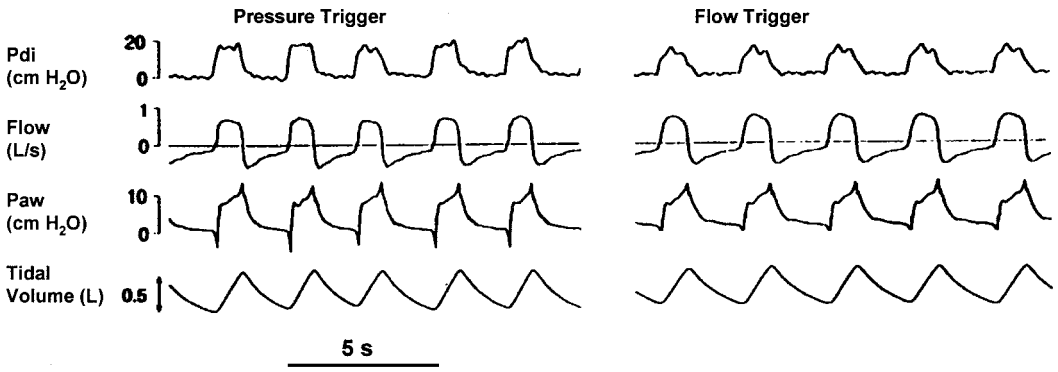

Fig. 2. Representative breaths during pressure-triggered and flow-triggered pressure support ventilation. The decrease in airway pressure (P_{aw}) during the trigger phase is smaller with flow-triggering. There is less muscle effort with flow-triggering, with no change in the P_{aw} contour. P_{di} = transdiaphragmatic pressure. (From Reference 10, with permission.)

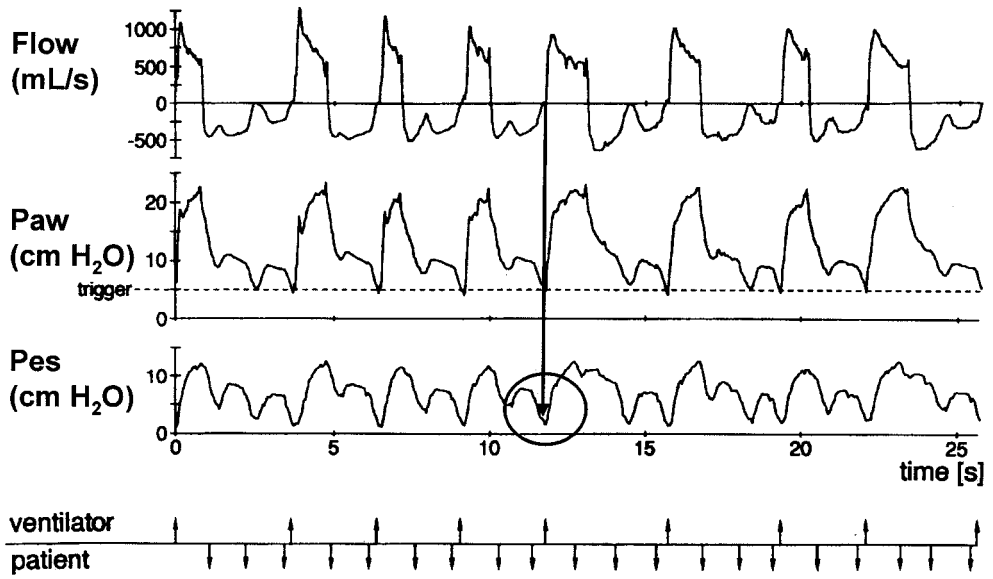

Fig. 3. Flow and pressure measured at the proximal airway (P_{aw}) and esophageal pressure (P_{es}) recorded from a patient receiving pressure support of 16 cm H_2O, positive end-expiratory pressure (PEEP) of 7 cm H_2O, trigger sensitivity of -2 cm H_2O. The down-pointing arrows represent patient efforts and the up-pointing arrows represent ventilator triggers. Note that the patient is breathing at 48 breaths/min but the ventilator is only triggering at 20 breaths/min. This patient's difficulty triggering the ventilator is due to intrinsic PEEP (auto-PEEP). Note that the patient must generate an inspiratory effort $>$ 5 cm H_2O to trigger the ventilator, suggesting an auto-PEEP of about 5 cm H_2O. When the patient's efforts are insufficient to overcome the level of auto-PEEP, the ventilator does not recognize the patient's effort. Note the effect of failed trigger efforts on the flow waveforms. (From Reference 16, with permission.)

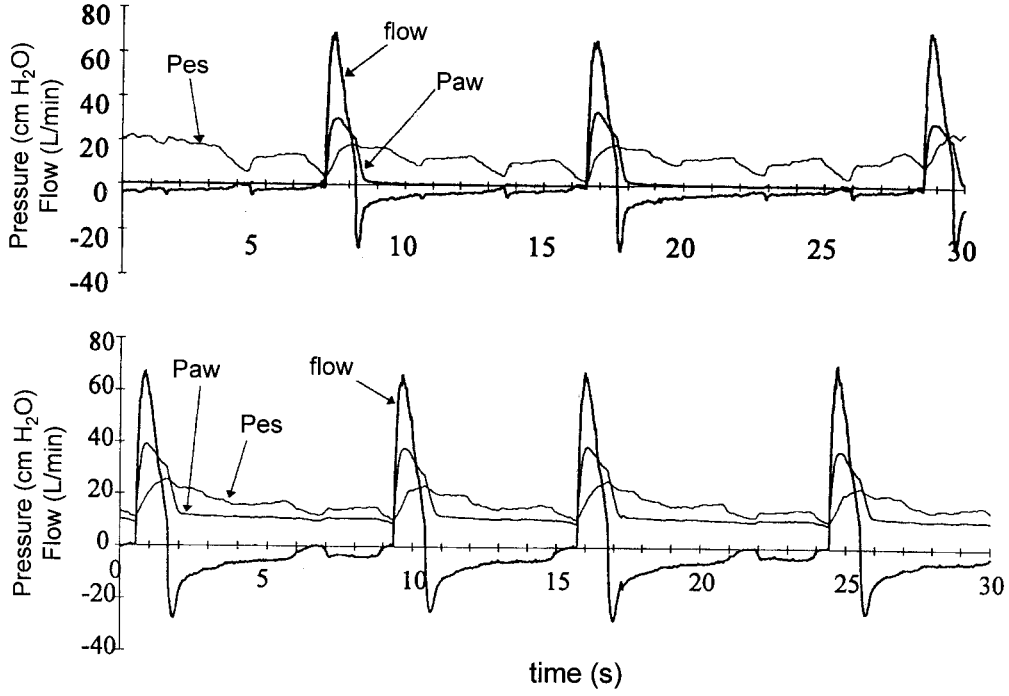

Fig. 4. Top panel: Esophageal pressure (P_{es}), airway pressure (P_{aw}), and flow at the tracheostomy. The patient's inspiratory efforts are identified by the negative P_{es} swings. The positive end-expiratory pressure (PEEP) is set at zero. P_{aw} appropriately drops to zero during expiration, demonstrating little circuit or valve resistance. Note that there is one triggered breath for every 3–4 efforts. Prolonged expiratory flow is due to airflow limitation. P_{es} swings have little effect in retarding the expiratory flow and even less effect on P_{aw}. Bottom panel: PEEP is increased to 10 cm H_2O. There is persistent flow at end-expiration; thus auto-PEEP is still present. Trigger dyssynchrony has improved, with 1 breath triggered every 2–3 inspiratory efforts. There is less limitation of expiratory flow, and the P_{es} swings are more effective in retarding the persistent expiratory flow. Note the effect of failed trigger efforts on the flow waveform. (From Reference 17, with permission.)

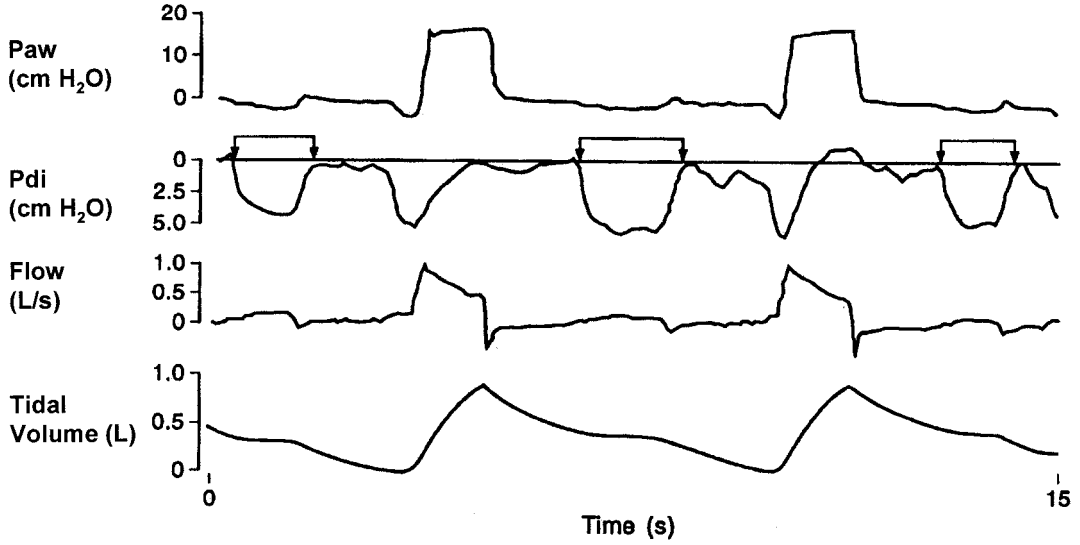

Fig. 5. Airway pressure (P_{aw}), transdiaphragmatic pressure (P_{di}), flow, and tidal volume during pressure support ventilation in a patient with chronic obstructive pulmonary disease (COPD). Note the presence of several ineffective efforts (between the arrows). (From Reference 18, with permission.)

phase. Most important, they found that the pressure triggers of current-generation ventilators are superior to those in older ventilators.

In patients with flow limitation, the presence of intrinsic positive end-expiratory pressure (auto-PEEP) is an important impediment to triggering.[11–13] To trigger the ventilator, the patient's effort must first overcome auto-PEEP before a pressure (or flow) change will occur at the proximal airway to trigger the ventilator. In those patients the effort to overcome auto-PEEP is much greater than the effort to trigger the ventilator. The addition of applied PEEP may counterbalance the auto-PEEP and improve the patient's ability to trigger. Several studies[14,15] have reported advantages of flow-triggering in patients with auto-PEEP, which is probably due to the base flow that causes a small increase in airway pressure due to resistance through the expiratory limb of the ventilation circuit. That increase in expiratory airway pressure counterbalances auto-PEEP and improves triggering.

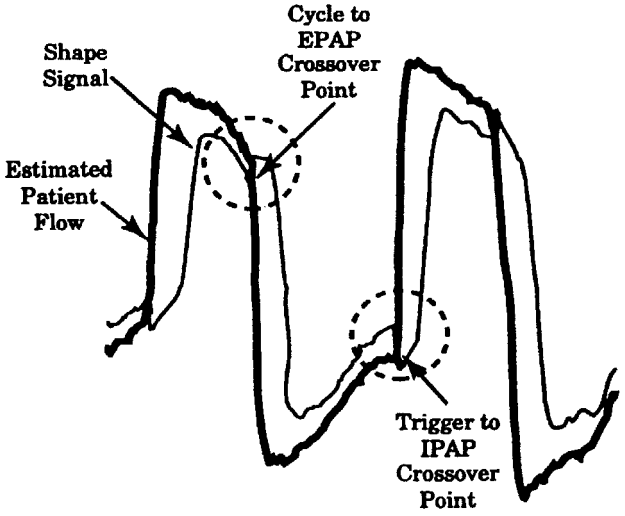

Fig. 6. Shape signal. A shape signal is produced by offsetting the actual patient flow signal by 15 L/min and delaying it by 300 ms. The intentional delay causes the ventilator-generated shape signal to be slightly behind the patient's flow rate. A sudden change in patient flow will cross the shape signal, causing the ventilator to trigger to the inspiratory phase or cycle to the expiratory phase. No evaluations of this have been reported. IPAP = inspiratory positive airway pressure. EPAP = expiratory positive airway pressure. (Courtesy of Respironics.)

Careful inspection of ventilator waveforms may allow detection of failed triggering efforts due to auto-PEEP. Fabry et al[16] observed trigger dyssynchrony in 9 of 11 patients recovering from acute respiratory failure with the application of PSV (Fig. 3). Chao et al[17] reported that trigger dyssynchrony commonly occurs in long-term mechanically ventilated patients, occurs more commonly in patients with a diagnosis of chronic obstructive pulmonary disease (COPD), and is associated with a poor outcome. Moreover, they found that adjusting the trigger sensitivity or changing from pressure triggering to flow triggering rarely affected the degree of trigger dyssynchrony. However, the addition of PEEP decreased the amount of (but often did not eliminate) trigger dyssynchrony. Those findings are consistent with auto-PEEP as the cause of trigger dyssynchrony (Fig. 4). Nava et al[18] reported that ineffective trigger efforts were likely in patients with COPD (Fig. 5). They also found that ineffective trigger efforts were

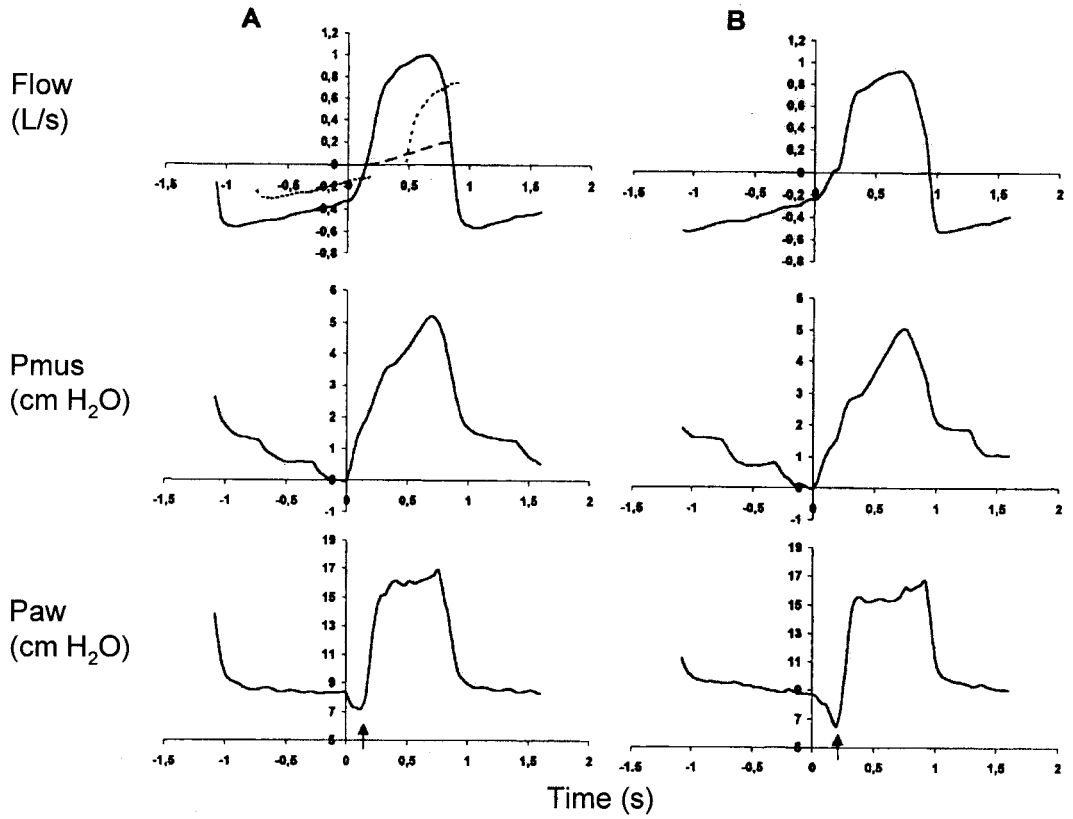

Fig. 7. Flow, respiratory muscle pressure (P_{mus}) and airway pressure (P_{aw}) as a function of time in a patient during pressure support ventilation delivered with (A) a Respironics Vision ventilator and (B) a Dräger Evita 4 ventilator. With the Vision ventilator the breath was triggered with the shape signal method. The beginning of neural inspiration was defined as zero time. Triggering of the ventilator (arrows) occurred 120 and 200 ms after the beginning of neural inspiration, respectively, with the Vision and Evita 4. Note that the drop in P_{aw} during the triggering phase was considerably less with the Vision that with the Evita 4. The better performance of the shape signal triggering method occurred despite the fact that with the Vision ventilator the expiratory flow at zero time was higher (0.33 vs 0.24 L/s), whereas inspiratory effort was comparable between ventilators. The dotted line represents the flow shape signal. The dashed line represents the electronic signal rising in proportion to actual inspiratory flow. (From Reference 19, with permission.)

reduced by PEEP that did not exceed the level of auto-PEEP.

A relatively new form of triggering is Auto-Trak, which is available with the Respironics Vision and S/T-D 30 ventilators. A shape signal is produced by offsetting the actual patient flow signal by 15 L/min and delaying it by 300 ms. This causes the ventilator-generated shape signal to be slightly behind the patient's flow rate (Fig. 6). A sudden change in patient flow will cross the shape signal, causing the ventilator to trigger to the inspiratory phase or cycle to the expiratory phase. Prinianakis et al[19] evaluated the effect of the shape-signal triggering method on patient-ventilator interaction during PSV. They compared triggering with the Respironics Vision ventilator, which used the shape signal, to the Dräger Evita 4 ventilator, which used flow-triggering at 2 L/min. They studied 12 patients and 3 levels of pressure support. They found that shape-signal triggering improved the ventilator function and decreased the patient effort during the triggering phase (Fig. 7). However, they also found that shape-signal triggering increased the number of auto-triggers.

One solution to triggering issues during PSV may be to couple the ventilator trigger to diaphragm electromyographic activity (Fig. 8).[20] In that way, ventilator triggering is tied to neural respiratory-center output. The neural trigger (ie, diaphragmatic electromyogram) is not affected by auto-PEEP and therefore does not require application of external PEEP for triggering purposes. When positive pressure is applied at the onset of diaphragmatic activity, the delay from the onset of inspiratory effort and mechanical assistance is shortened, the esophageal pressure deflection is reduced, and the WOB is decreased. In situations where conventional triggering cannot provide ventilator support in synchrony with the patient's neural inspiratory drive, neural triggering has the potential to improve patient-ventilator

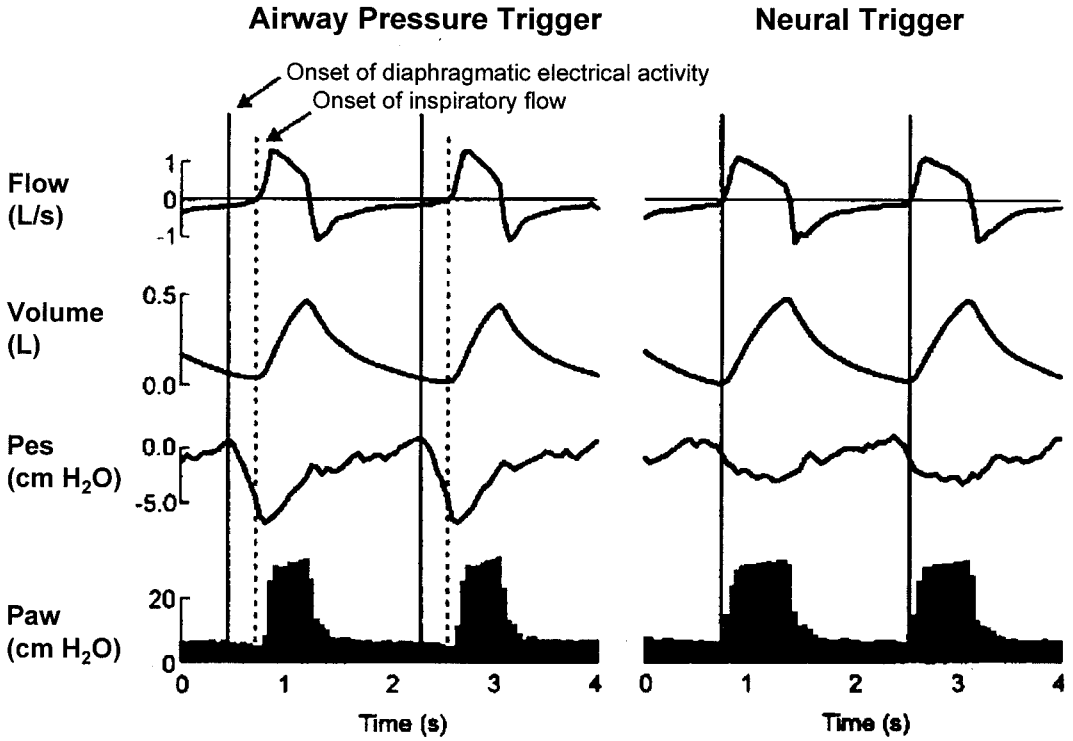

Fig. 8. With a conventional pressure trigger (left), mechanical ventilatory support starts when airway pressure decreases by a preset amount. The beginning of inspiratory effort (solid vertical line) precedes inspiratory flow by several hundred milliseconds. That delay is due to intrinsic positive end-expiratory pressure and occurs despite externally applied PEEP. There is also a further delay from the onset of inspiratory flow (vertical dashed line) to the rise in positive airway pressure, due to the mechanical limitation of the ventilator trigger. With neural triggering (right), the ventilator provides support as soon as diaphragmatic electrical activity exceeds a threshold level. The delay to onset of inspiratory flow and increase in airway pressure is almost eliminated. P_{es} = esophageal pressure. P_{aw} = airway pressure. (From Reference 20, with permission.)

interaction. However, this approach is investigational at present and its clinical usefulness remains to be determined.

More sensitive settings may result in auto-triggering due to signal noise, such as leaks, patient movement, water in the ventilator circuit, and cardiac oscillations. Imanaka et al[21] found that auto-triggering caused by cardiogenic oscillation was common in post-cardiac-surgery patients when flow-triggering was used (Fig. 9). Auto-triggering occurred more often in patients with more dynamic circulation and caused respiratory alkalosis and hyperinflation of the lungs.

The lack of a backup rate with pressure support may be problematic. Parthasarathy and Tobin[22] evaluated the effect of ventilation mode on sleep quality among 11 critically ill patients. Sleep fragmentation was greater during PSV than during continuous mandatory ventilation (Fig. 10). Central apneas occurred during PSV in 6 patients, and heart failure was more common in those 6 patients than in the 5 patients without apneas. Changes in sleep-wakefulness state caused greater changes in end-tidal P_{CO_2} during PSV than during continuous mandatory ventilation. The authors concluded that PSV causes hypocapnia, which, combined with the lack of a backup rate and wakefulness drive, can lead to central apneas and sleep fragmentation, especially in patients with heart failure.

The Equation of Motion As It Applies to PSV

The interactions between the ventilator and the patient can be described by the equation of motion, which states that the pressure required to deliver a volume of gas into the lungs is determined by the elastic and resistive properties of the respiratory system. With PSV the pressure is the sum of the pressure that the ventilator applies to the airway (P_{aw}) and the pressure generated by the respiratory muscles (P_{mus}). The elastic properties of the respiratory system are determined by compliance (C) and tidal volume (V_T), and the resistive properties of the lungs are determined by flow ($\dot{V}$) and airways resistance (R). The equation of motion thus becomes:

$$P_{aw} + P_{mus} = V_T/C + \dot{V} \times R \quad (1)$$

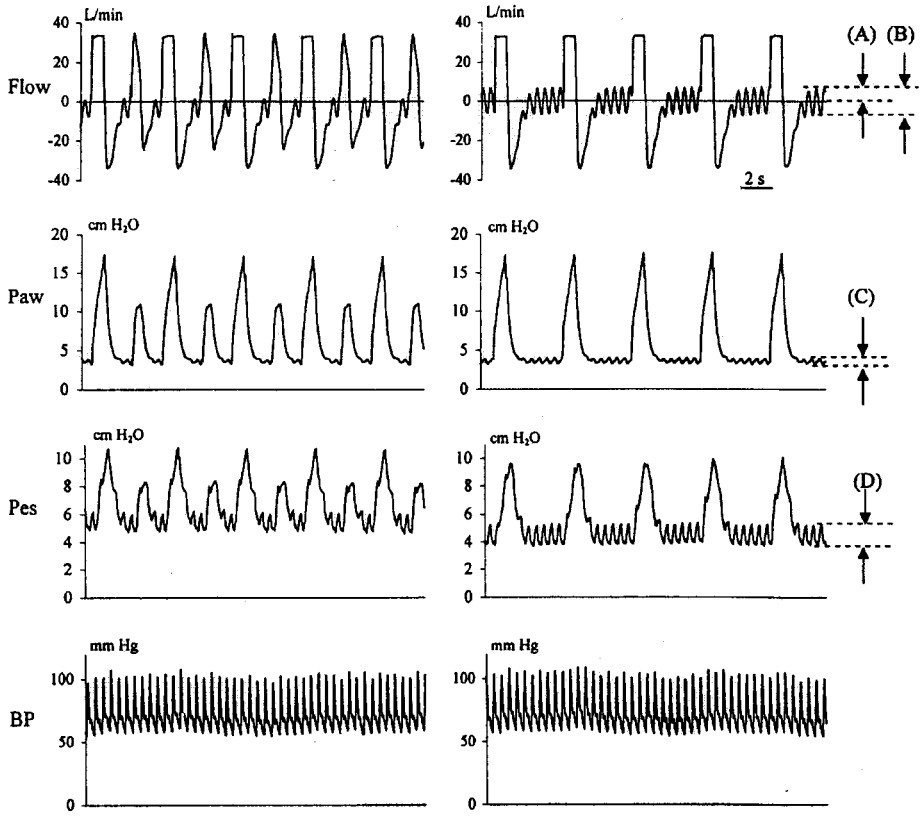

Fig. 9. Flow, volume, esophageal pressure (P_{es}), airway pressure (P_{aw}), and blood pressure (BP) waveforms from a patient who underwent mitral valve replacement and tricuspid annuloplasty for mitral stenosis, tricuspid regurgitation, and aortic regurgitation. With triggering sensitivity set at 1 L/min (left), pressure support ventilation was activated between 2 synchronized intermittent-mandatory-ventilation breaths. When trigger sensitivity was changed to 4 L/min (right), pressure support breaths disappeared and there were marked oscillations in flow, P_{aw}, and P_{es}. Cardiogenic oscillation was evaluated as the peak inspiratory flow fluctuation (A), amplitude in the flow oscillation (B), amplitude in airway pressure (C), and amplitude in esophageal pressure (D). Also note that the baseline P_{es} was elevated when autotriggering occurred, suggesting hyperinflation of the lungs. (From Reference 21, with permission.)

During PSV, P_{aw} is fixed by the ventilator. If the patient generates an inspiratory effort during PSV (ie, greater P_{mus}), flow and volume delivery increase. P_{mus} is determined by respiratory drive and respiratory muscle strength.

Note that an increase in pressure support will not affect flow and V_T if there is a subsequent decrease in respiratory drive, which results in a lower P_{mus}. Changes in pressure support might result in one of several effects on V_T. If P_{mus} remains constant when pressure support is changed, then the V_T will change. However, if P_{mus} changes in response to the change in pressure support, then the V_T might not change. For example, an increase in pressure support may unload respiratory muscles, producing a decrease in P_{mus} and little change in V_T (Fig. 11).

Muscle Pressure

The time course of P_{mus} can be approximated by the second-order polynomial function:[23,24]

$$P_{mus}(t) = -d \times (t \times Ti)^2 + d \times Ti^2 \quad (2)$$

in which d is a constant, and Ti is defined as the time between the onset of the increase in inspiratory P_{mus} and the start of its decline. It is assumed that P_{mus} reaches its maximum and becomes flattened at the end of the neural inspiratory effort. Thus, the maximum $P_{mus-max}$ can be expressed as:

$$P_{mus-max} = d \times Ti^2 \quad (3)$$

Substituting into the above equation yields:

$$P_{mus}(t) = -P_{mus-max} \times (1 - t/Ti)^2 + P_{mus-max} \quad (4)$$

in which $0 \leq t \leq Ti$. This is illustrated in Figure 12.

Airway Pressure During PSV

Figure 13 shows a schematized airway pressure waveform during PSV.[25] The initial airway-pressure change

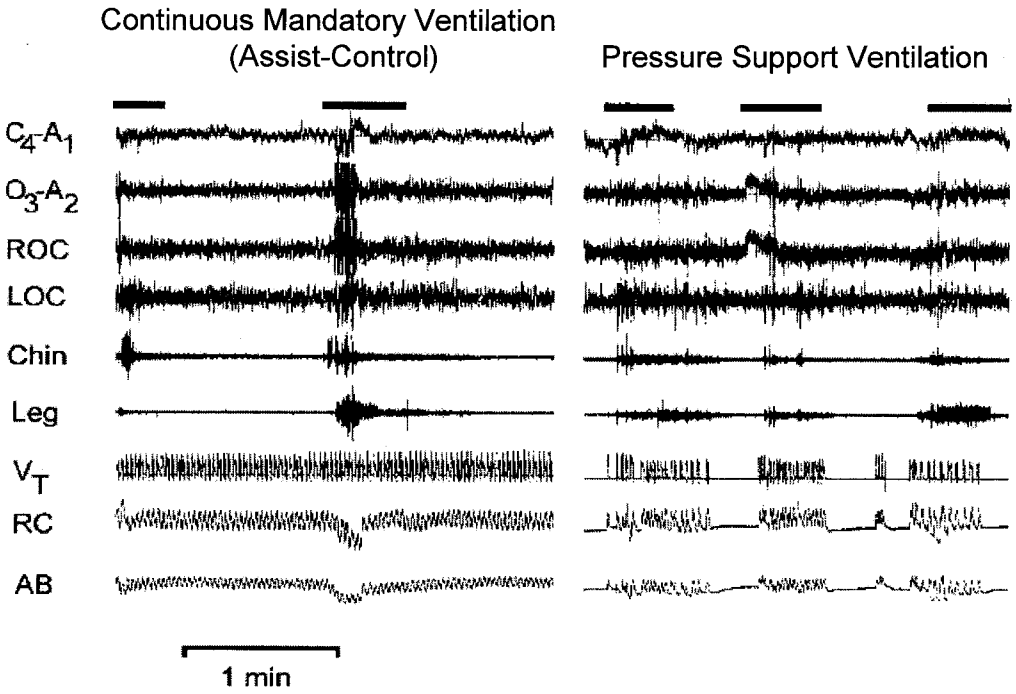

Fig. 10. Polysomnography waveforms during assist-control ventilation and pressure support of a representative patient. From top to bottom, the waveforms show electroencephalogram (C4-A1, O3-A2), electrooculogram (ROC, LOC), electromyograms (chin and leg), integrated tidal volume (V_T), rib-cage (RC), and abdominal (AB) excursions on respiratory inductive plethysmography. Arousals and awakenings, indicated by horizontal bars, were more numerous during pressure support than during continuous mandatory ventilation (assist-control). (From Reference 22, with permission.)

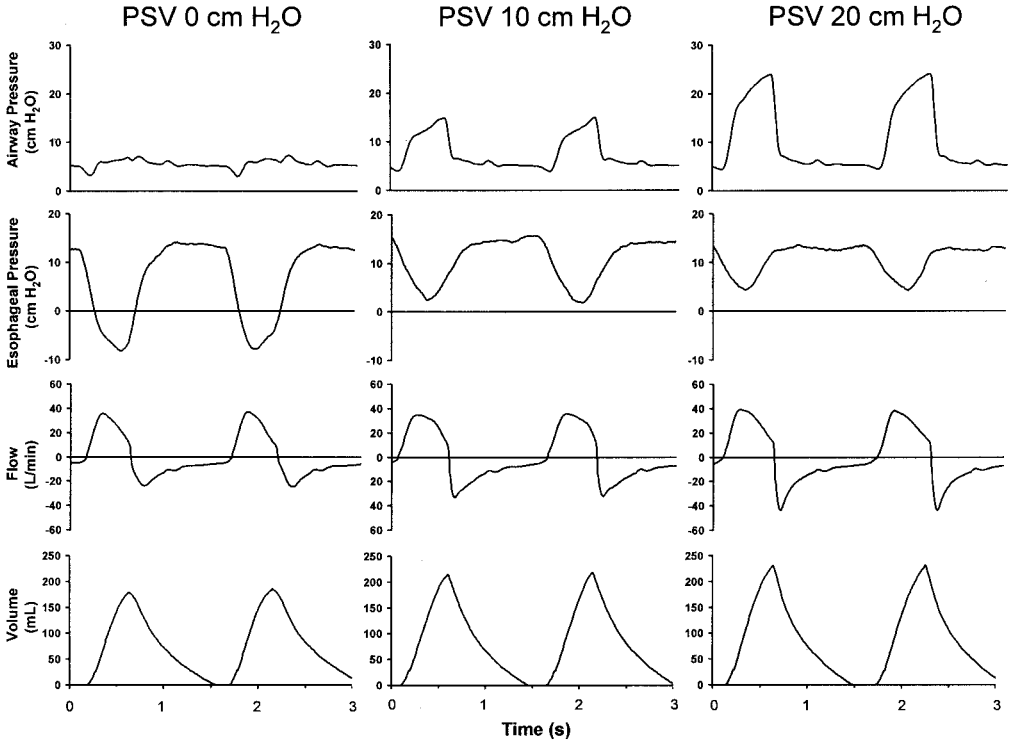

Fig. 11. Airway pressure, esophageal pressure, flow, and tidal volume in a patient with 0, 10, and 20 cm H_2O of pressure applied to the airway. Note the decrease in esophageal pressure as airway pressure is increased. There is only a small increase in tidal volume with the increase in pressure support. In this case the principal effect of pressure support is to provide respiratory muscle unloading. PSV = pressure support ventilation.

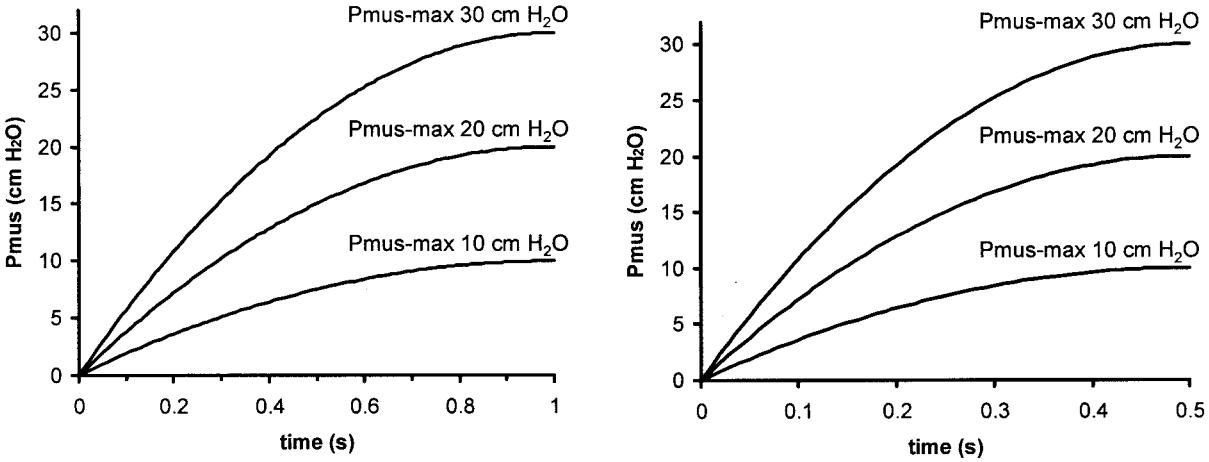

Fig. 12. Changes in the pressure generated by the respiratory muscles (P_{mus}) with 3 levels of maximum P_{mus} ($P_{mus-max}$) and 2 levels of neural inspiratory time. Note that a higher $P_{mus-max}$ and a shorter neural inspiratory time translate clinically into a greater respiratory drive.

during PSV can be described mathematically. Once the ventilator is triggered, P_{aw} increases exponentially to the pressure support level with a ventilator time constant (τv) and then stays at that level until the termination of the inspiratory phase:[24]

$$P_{aw}(t) = P_{PS} \times (1 - e^{-t/\tau v}) \quad (5)$$

in which P_{PS} is the pressure support setting, e is the base of the natural logarithm, and $t \geq 0$. Figure 14 shows airway pressure waveforms for several levels of P_{PS} and τv.

In previous generations of ventilators, τv was preset in the engineering of the ventilator. Many current-generation ventilators allow the clinician to adjust τv. From

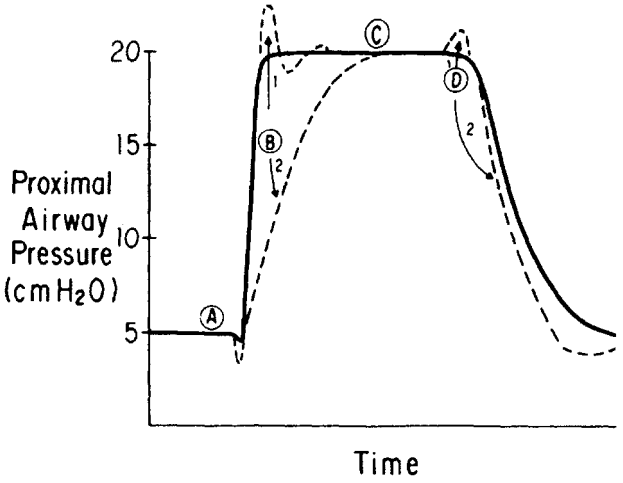

Fig. 13. Characteristics of a pressure supported breath. In this example, baseline pressure (ie, positive end-expiratory pressure [PEEP]) is set at 5 cm H_2O and pressure support is set at 15 cm H_2O. The inspiratory pressure is triggered at point A by a patient effort, resulting in an airway pressure decrease. The rise to pressure (line B) is provided by the initial flow into the airway. If the initial flow is excessive, initial pressure exceeds set level (B1). If the initial flow is low, a slow rise to pressure occurs (B2). The plateau of pressure support (line C) is maintained by control of flow. A smooth plateau indicates appropriate flow responsiveness to patient demand. Termination of pressure support occurs at point D and should coincide with the end of neural inspiration. If breath termination is delayed, the patient may actively exhale (rise in pressure above plateau) (D1). If breath termination is premature, the patient may have continued inspiratory efforts (D2). (From Reference 25, with permission.)

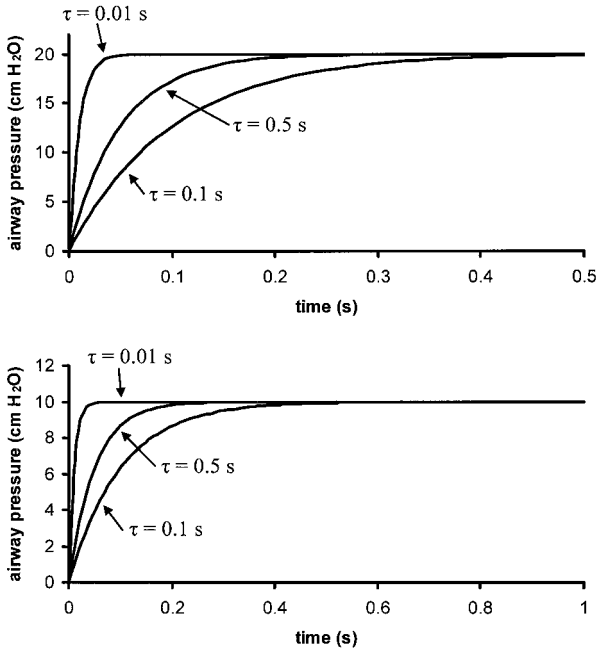

Fig. 14. The effect of rise time (τ) on the pressurization rate at the initiation of the inspiratory phase. Illustrated are 2 pressure support levels: 20 cm H_2O (upper panel) and 10 cm H_2O (lower panel).

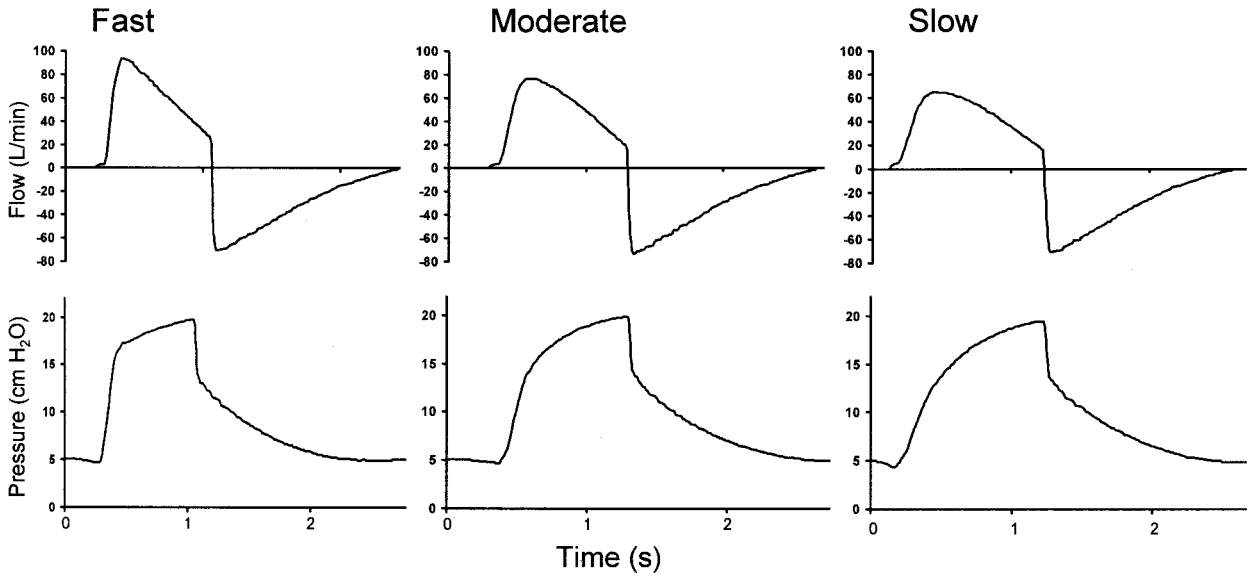

Fig. 15. Flow and pressure waveforms for 3 rise times (pressurization rates) at a pressure support of 20 cm H_2O. Note the effect of rise time on flow at the initiation of the inspiratory phase.

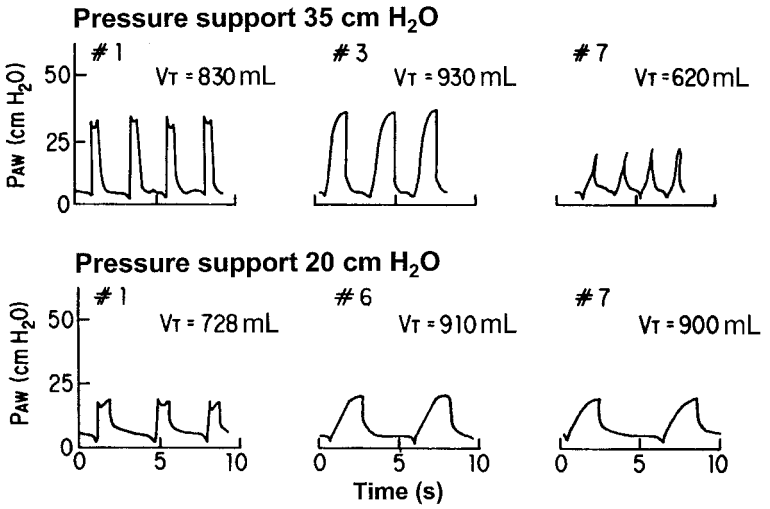

Fig. 16. Examples of airway pressure waveforms from 2 patients using different rise times during pressure support ventilation. The maximum rise time is with the waveforms labeled #1. The minimum rise time is with the waveforms labeled #7. Note that the optimal rise time for the first patient (top 3 waveforms) is at a high setting, whereas the optimal rise time for the second patient (bottom 3 waveforms) is at a slow setting. P_{aw} = airway pressure. V_T = tidal volume. (From Reference 26, with permission.)

an operational standpoint, this becomes the rise-time setting on the ventilator. The term "rise time" refers to the time required for the ventilator to reach the pressure support setting at the onset of inspiration; it is the rate of pressurization at the initiation of the inspiratory phase. The rise time should be adjusted to patient comfort, and ventilator waveforms may be useful to guide this setting. The rise-time adjustment effectively allows the clinician to set the flow at the onset of the inspiratory phase during PSV. Note that a fast rise time (one in which the ventilator reaches the pressure support setting quickly) is associated with high flow at the onset of inhalation (Fig. 15). On the other hand, a slow rise time (one in which the ventilator reaches the pressure support setting slowly) is associated with a lower flow at the onset of inhalation. Theoretically, patients with a high respiratory drive should benefit from a fast rise time, whereas those with a lower respiratory drive might benefit from a slower rise time.

MacIntyre and Ho[26] found that the optimal rise time for some patients is at a high setting, whereas the optimal rise time for others is at the slow setting (Fig. 16).

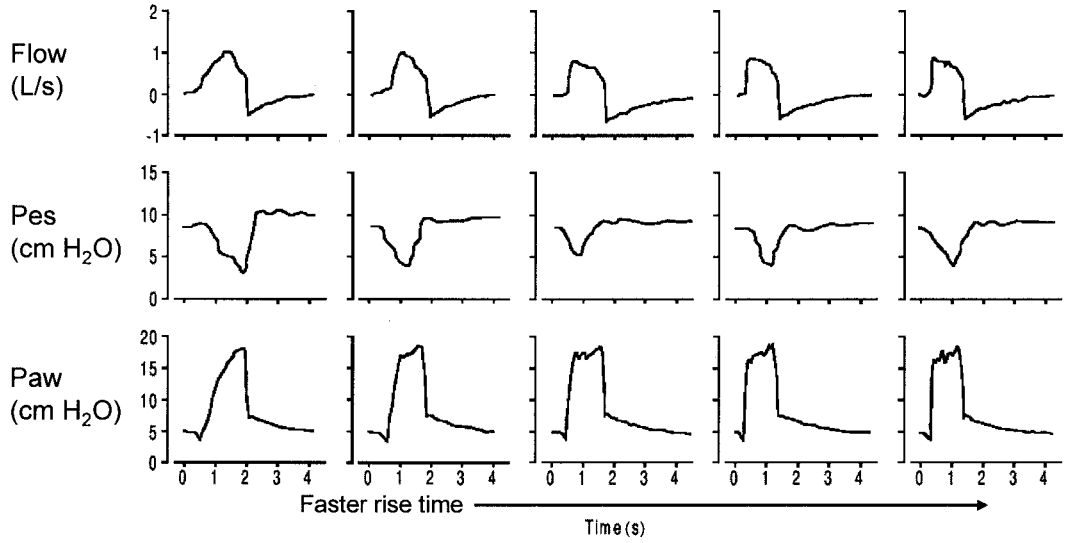

Fig. 17. An example of flow, esophageal pressure (P_{es}), and airway pressure (P_{aw}) at 5 different rise times. Note the effect of rise-time setting on flow and P_{es} swing. Also note that the lowest P_{es}-swing occurs at the intermediate rise-time setting. (From Reference 32, with permission.)

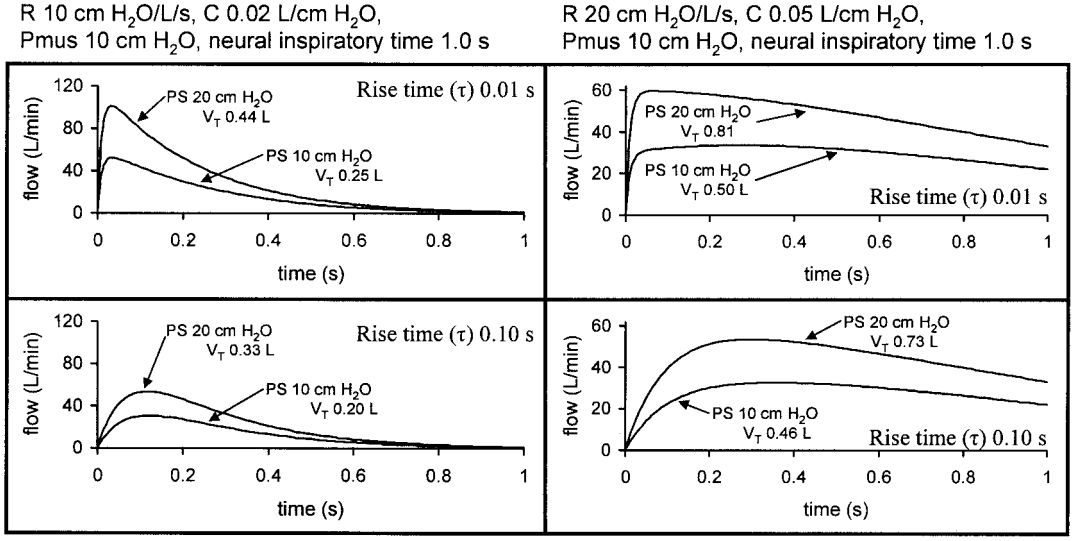

Fig. 18. Airway-flow waveforms during pressure support ventilation with 2 types of lung mechanics. The waveforms on the left illustrate the conditions of restrictive lung disease (eg, acute lung injury) and those on the right illustrate conditions of obstructive lung disease. Also illustrated are 3 levels of pressure support and 2 rise times. Note that flow at the beginning of inhalation increases with a faster rise time and higher pressure support setting, with either set of lung mechanics. R = resistance. C = compliance. P_{mus} = pressure generated by the respiratory muscles. PS = pressure support.

Branson et al[27] also found that individual patient titration of the rise time was necessary to optimize the efficacy of PSV. Bonmarchand et al[28,29] reported the lowest work of breathing (WOB) with the fastest rise time (ie, the one that reached the pressure support setting in 0.1 s, compared to 0.5 s, 1 s, and 1.5 s) in patients with restrictive lung disease and COPD. Mancebo et al[30] reported that a slower rise time increased the WOB, although it did not affect the V_T and respiratory rate. In patients with a high respiratory drive, Uchiyama et al[31] reported that a fast rise time was as effective as increasing the level of pressure support (note that either a faster rise or higher pressure support setting will increase the flow at the onset of inhalation). Chiumello et al[32] studied the effects of different rise times during PSV on breathing pattern, WOB, gas exchange and patient comfort in patients with acute lung injury. They found that the lowest pressurization rate (slow pressure

Table 1. Cycle Criteria for Some Commonly Used Adult Mechanical Ventilators

Ventilator	Flow Cycle	Pressure Cycle	Time Cycle
Puritan-Bennett 7200	5 L/min	PEEP + pressure support + 1.5 cm H_2O	3 s
Puritan-Bennett 840	Adjustable (1–80% of peak flow)	PEEP + pressure support + 1.5 cm H_2O	3 s
Puritan-Bennett 740/760	10 L/min or 25% of peak flow	PEEP + pressure support + 3 cm H_2O	3.5 s
Servo 900C	25% of peak flow	PEEP + pressure support + 3 cm H_2O	80% of set cycle time
Servo 300	5% of peak flow	PEEP + pressure support + 20 cm H_2O	80% of set cycle time
Servo*i*	Adjustable (1–40% of peak flow)	High-pressure limit	≤ 2.5 s, based on flow-cycle setting*
Dräger Evita 4	25% of peak flow	High-pressure limit	4 s
Bear 1000	25% of peak flow	High-pressure limit	5 s
Hamilton Veolar	25% of peak flow	High-pressure limit	3 s
Hamilton Galileo	Adjustable (10–40% of peak flow)	High-pressure limit	3 s
Infrasonics Star	4 L/min	PEEP + pressure support + 3 cm H_2O	3.5 s
Bird 8400 and TBird	25% of peak flow	High-pressure limit	3 s
Pulmonetic LTV	Adjustable (10–40% of peak flow)	High-pressure limit	Adjustable (1–3 s)
Viasys Avea	Adjustable (5–45% of peak flow)	High-pressure limit	Adjustable (0.2–5.0 s)
Newport E500	Variable, based on time constant and pressure above pressure support setting	High-pressure limit	3 s

PEEP = positive end-expiratory pressure
*Flow drops to a range between 25% of peak flow and flow-cycle criteria, and time in this range exceeds 50% of the time before entering this range.

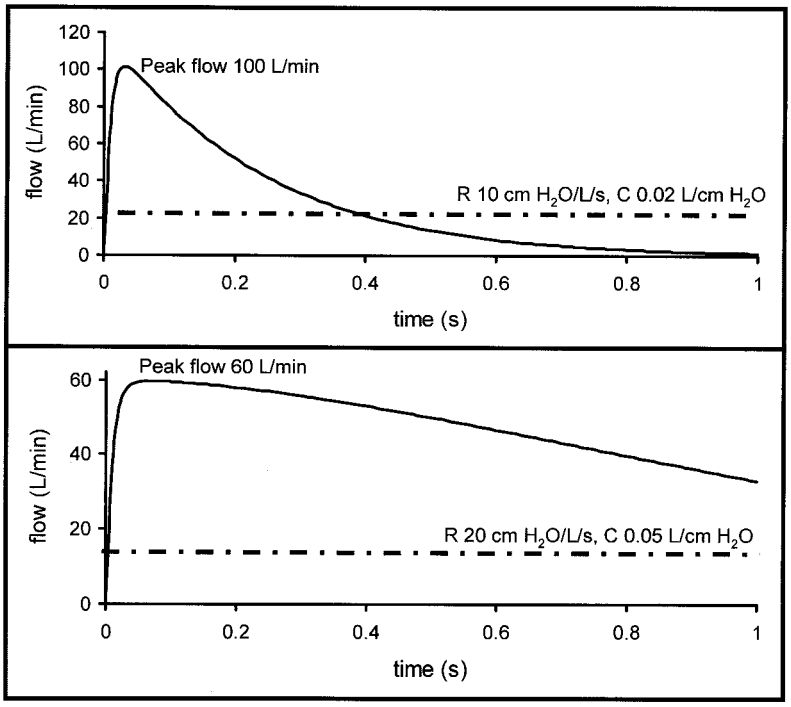

Fig. 19. Effect on respiratory mechanics of cycling of pressure support from inhalation to exhalation. Pressure support is set at 20 cm H_2O, rise time (τ) is 0.01 s, and the maximum pressure generated by the respiratory muscles ($P_{mus-max}$) is 10 cm H_2O. Flow-termination is set at 25% of the peak pressure, as illustrated by the broken line. The upper panel represents the respiratory mechanics of a patient with restrictive lung disease. The lower panel represents the respiratory mechanics of a patient with obstructive lung disease. In each case, the neural inspiratory time is 1.0 s. Note that the breath terminates prematurely in the patient with restrictive lung disease, but the breath is prolonged in the patient with obstructive lung disease. Also note that the peak flow is greater in restrictive lung disease and the pressure decrease is more rapid in restrictive lung disease. R = resistance. C = compliance.

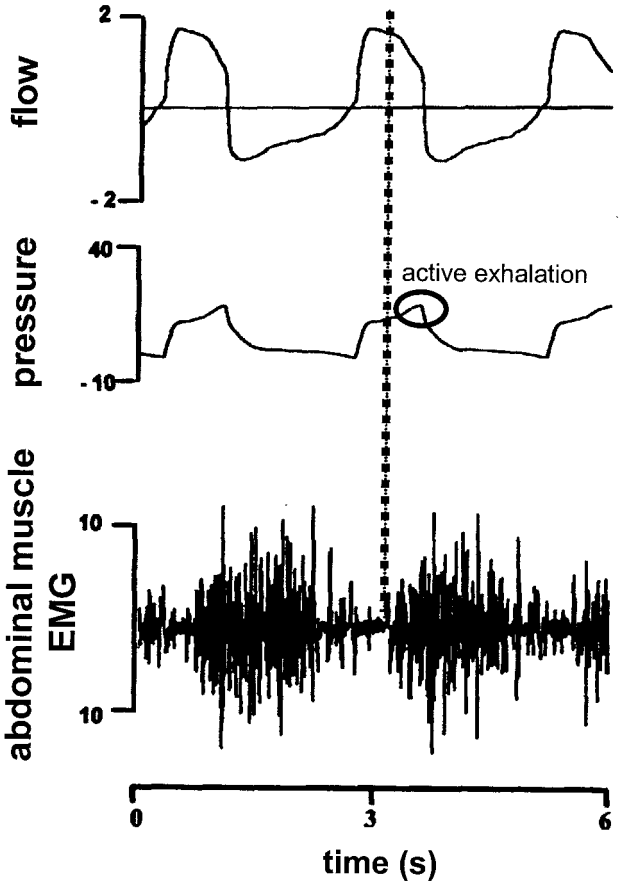

Fig. 20. Flow, airway pressure, and transversus abdominis electromyogram (EMG) from a mechanically ventilated patient with chronic obstructive pulmonary disease receiving pressure support ventilation at 20 cm H_2O. The onset of expiratory muscle activity (vertical dotted line) occurred when mechanical inflation was only partly completed, as indicated by the onset of expiratory muscle activity. Also note that active exhalation causes an increase in airway pressure at end-exhalation, causing the ventilator to pressure-cycle rather than flow-cycle. (From Reference 41, with permission.)

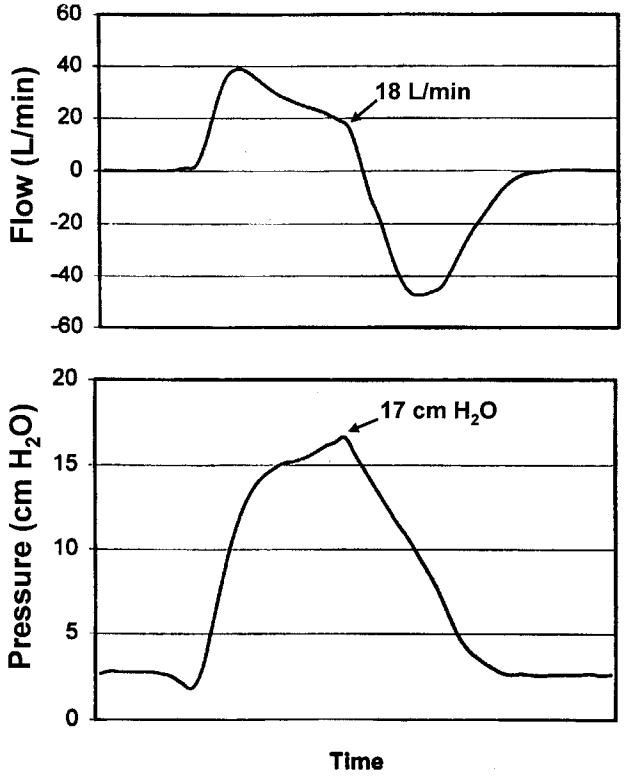

Fig. 21. An example of delayed termination of inhalation during pressure support ventilation in a patient with chronic obstructive pulmonary disease. The patient was ventilated with a Puritan-Bennett 7200 ventilator, using pressure support ventilation at 12 cm H_2O and positive end-expiratory pressure of 3 cm H_2O (inspiratory pressure of 15 cm H_2O), which has a flow termination of 5 L/min during pressure support. Note that the ventilator cycles at a flow of 18 L/min. The pressure increase above the set level of pressure support causes the ventilator to pressure-cycle in response to the patient's active exhalation. (From Reference 42, with permission.)

rise) caused the lowest V_T, highest respiratory rate, and highest WOB (Fig. 17). The other pressurization rates produced no differences in breathing pattern or WOB. Patient comfort was worse at the lowest and highest pressurization rates. In patients with COPD recovering from acute hypercapnic respiratory failure and receiving noninvasive positive-pressure ventilation, Prinianakis et al[33] reported that the greatest reduction in the pressure-time product of the diaphragm occurred with the highest pressurization rate (fast pressure rise), but that was accompanied by substantial air leaks and poor tolerance.

There are several potential drawbacks to a high inspiratory flow at the onset of inspiration (such as might result from a higher pressurization rate).[34] First, if the flow is higher at the onset of inspiration, the inspiratory phase may be prematurely terminated if the ventilator cycles to the expiratory phase at a flow that is a fraction of the peak inspiratory flow. Second, several studies have suggested the existence of a flow-related inspiratory terminating reflex.[35–39] Activation of this reflex causes a shortening of neural inspiration, which could result in brief, shallow inspiratory efforts (particularly at low pressure support settings). The clinical effects of this inspiratory-flow-terminating reflex during PSV remains to be determined. At the least, it suggests that manipulation of rise time during PSV may result in a complex interaction between ventilator function and physiology.

Flow and Volume Delivery During PSV

During PSV, inspiratory flow is determined by the pressure applied to the airway (ie, pressure support setting), the pressure generated by the respiratory muscles (P_{mus}), the airways resistance, and the time constant:

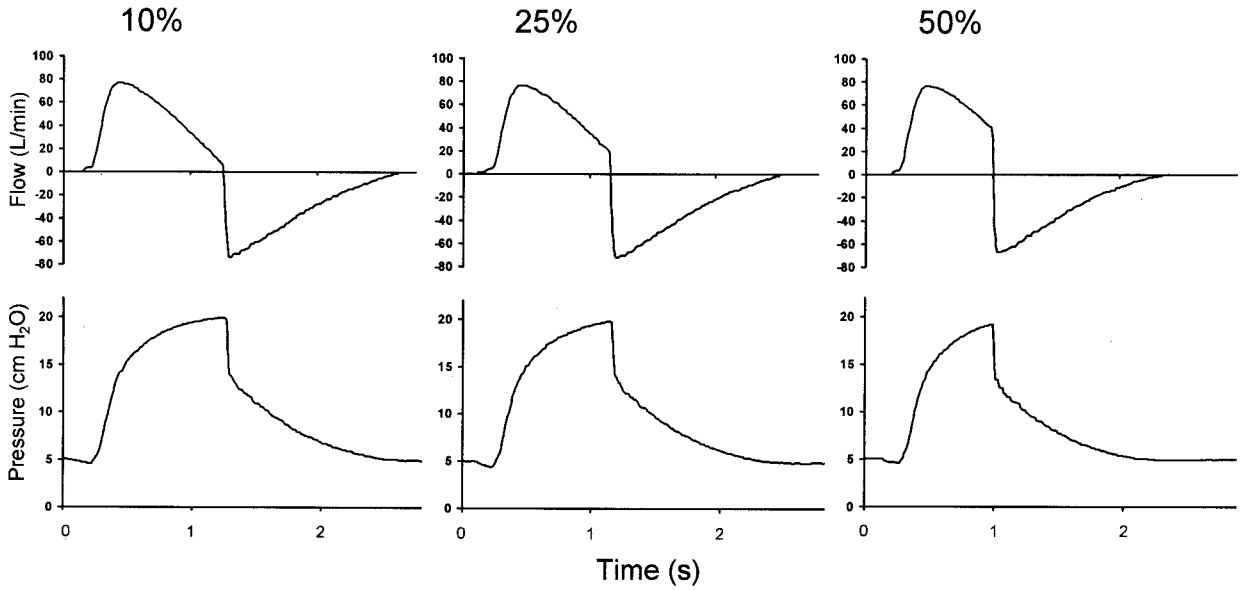

Fig. 22. Examples of flow-termination criteria of 10%, 25%, and 50%, using a Puritan-Bennett 840 ventilator with pressure support 15 cm H_2O and positive end-expiratory pressure of 5 cm H_2O. Lung model settings were: resistance 5 cm H_2O/L/s, compliance 0.05 L/cm H_2O.

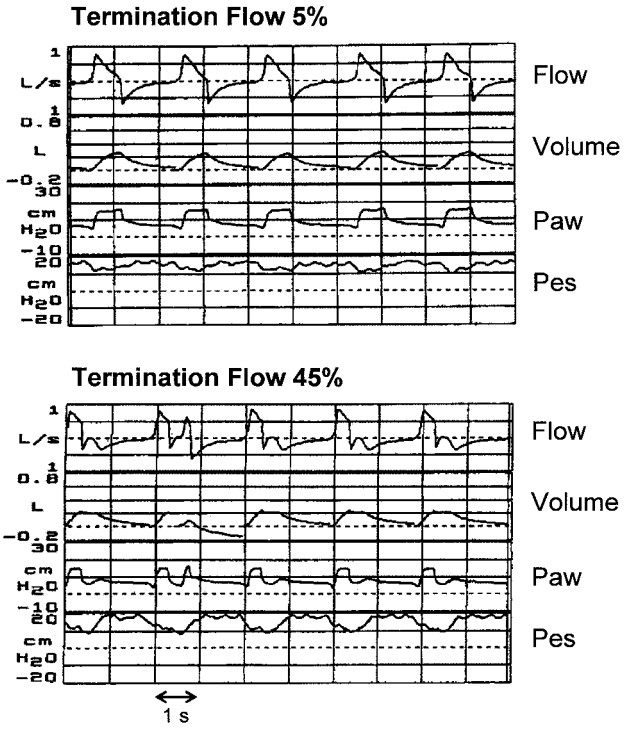

Fig. 23. Flow, volume, airway pressure (P_{aw}), and esophageal pressure (P_{es}) waveforms with flow-termination criteria of 5% and 45%. With flow termination of 5%, inspiratory flow terminated simultaneously with the cessation of inspiratory effort, estimated by P_{es}. In contrast, premature termination with double-triggering occurred with flow termination of 45%. (From Reference 46, with permission.)

$$\dot{V} = (\Delta P/R) \times (e^{-t/\tau}) \quad (6)$$

in which ΔP is the sum of pressure support and P_{mus}, R is airways resistance, e is the base of the natural logarithm, t is the elapsed time after initiation of the inspiratory phase, and τ is the product of airways resistance and respiratory system compliance (the time constant of the respiratory system). This is illustrated in Figure 18.

The area of the flow-time curve is the delivered V_T:

$$V_T = \int \dot{V}\, dt \quad (7)$$

Thus, V_T during PSV is determined primarily by the pressure support setting, the inspiratory effort of the patient, airways resistance, respiratory-system compliance, and inspiratory time. The delivered V_T will also be affected by auto-PEEP. An increase in auto-PEEP effectively decreases the driving pressure gradient, and thus the V_T decreases. Theoretically, the delivered V_T will be zero if the auto-PEEP equals the pressure support setting. Thus, auto-PEEP will affect breath delivery in 2 ways during PSV. First, it increases the effort required to trigger the ventilator. Second, it decreases the delivered V_T.

The Cycle From Inhalation to Exhalation

During PSV, the ventilator is normally flow-cycled. The flow at which the ventilator cycles can be a fixed absolute

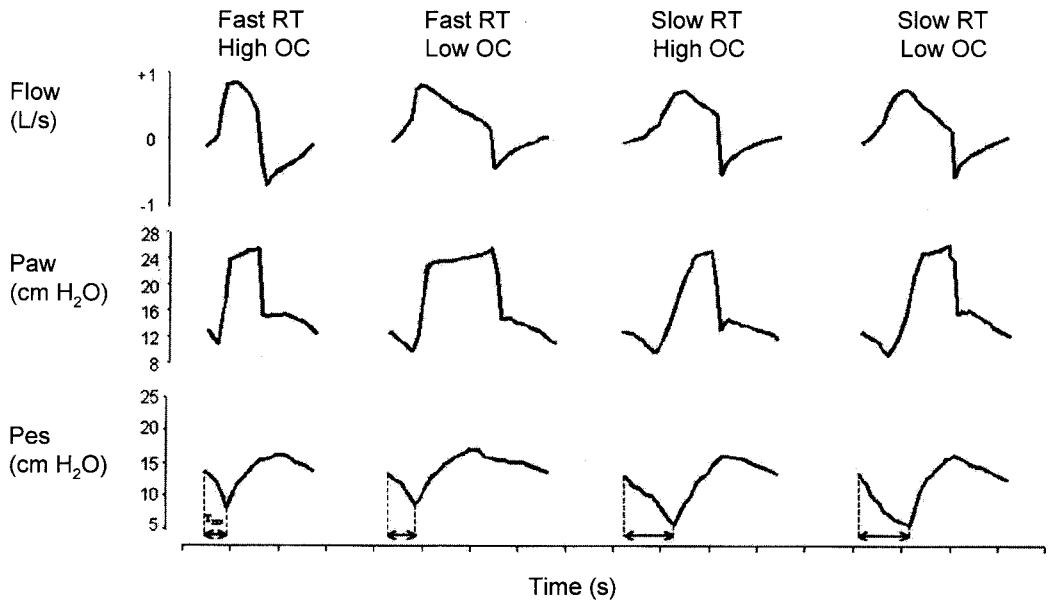

Fig. 24. Examples of flow, airway pressure (P_{aw}), and esophageal pressure (P_{es}) waveforms at various inspiratory rise-time and flow-cycle criteria combinations. RT = rise time. OC = off criteria (the flow that terminates the inspiratory phase). (From Reference 47, with permission.)

Table 2. Effect of Pressure Support Setting, P_{mus}, and Time Constant on the Appropriate Flow-Termination Setting During Pressure Support Ventilation*

Resistance (cm H_2O/L/s)	Compliance (L/cm H_2O)	τ (s)	10 cm H_2O $P_{mus-max}$			30 cm H_2O $P_{mus-max}$		
			PS 10	PS 20	PS 30	PS 10	PS 20	PS 30
20	0.8	1.14	79	70	65	85	82	79
20	0.4	0.66	58	48	43	70	64	58
20	0.2	0.36	30	22	19	44	36	30
5	0.8	0.29	21	15	12	34	26	21
5	0.4	0.17	8	5	4	16	11	8
5	0.2	0.09	3	2	1	6	4	3

PS = pressure support setting
*Note that the flow-termination ranges from as low as 1% of peak flow to as high as 85% of peak flow.
$P_{mus-max}$ = maximum pressure generated by the respiratory muscles
τ = time constant
(Data from Reference 24.)

flow, a flow based on the peak inspiratory flow, or a flow based on peak inspiratory flow and elapsed inspiratory time (Table 1). In some cases, the cycle is quite sophisticated. The Respironics Vision ventilator, for example, uses the shape signal to cycle the ventilator (see Fig. 6).

Ideally, the ventilator should cycle to exhalation at the end of the neural inspiratory time. If the breath terminates before the end of neural inhalation, the patient may double-trigger the ventilator. If breath delivery continues into neural exhalation, the patient may actively exhale, causing the ventilator to pressure-cycle rather than flow-cycle. The inspiratory time during PSV is determined by lung mechanics and the flow cycle criteria (Fig. 19).

Several studies have reported dyssynchrony with PSV in subjects who have airflow obstruction (eg, COPD).[40–42] With airflow obstruction the inspiratory flow decreases slowly, the flow cycle criteria may not be reached at the end of neural inhalation, and this stimulates active exhalation to pressure-cycle the breath (Figs. 20 and 21). This can be seen on the ventilator waveforms as a rise in pressure at end-exhalation that exceeds the pressure support setting on the ventilator. This problem increases with higher levels of pressure support and with higher levels of airflow obstruction. Mathematical and laboratory analyses by Hotchkiss et al[43–45] showed that PSV in the setting of airflow obstruction can be accompanied by marked variations in

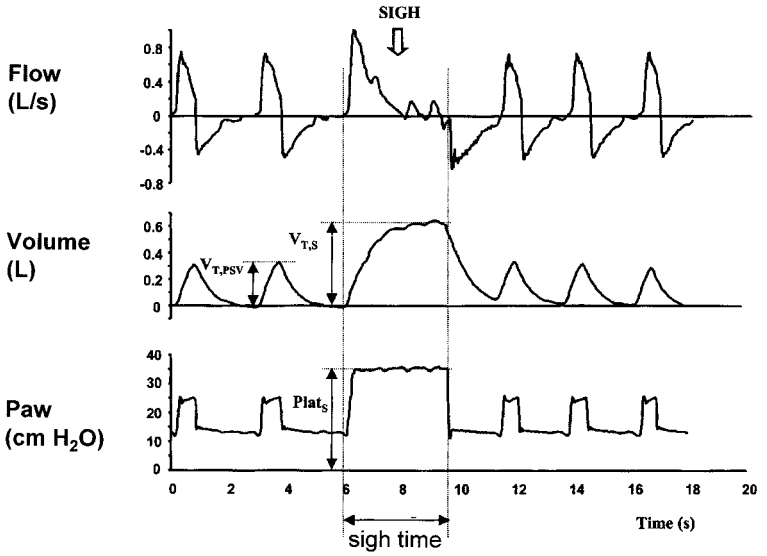

Fig. 25. An example of the waveforms from use of a sigh breath in conjunction with pressure support ventilation. The patient was ventilated with a Dräger Evita 4 ventilator (in PCV+ mode). P_{aw} = airway pressure. (From Reference 51, with permission.)

V_T and auto-PEEP, even when the subject's effort is unvarying. The mechanism underlying this observed instability is "feed forward" behavior mediated by oscillatory elevations in auto-PEEP. Approaches to correct this problem during PSV include: (1) administer bronchodilators and clear secretions to decrease airways resistance, (2) use a lower level of pressure support, (3) use pressure-controlled ventilation with the inspiratory time set short enough that the patient does not contract the expiratory muscles to terminate inspiration (eg, 0.8–1.2 s) or the inspiratory time can be adjusted by observing patient comfort and avoiding a period of zero flow at the end of inspiration, (4) adjust the flow at which the ventilator cycles (Fig. 22).

Several studies also examined flow termination during PSV in patients recovering from acute lung injury. Tokioka et al[46] reported that higher levels of flow termination in that patient population resulted in a lower V_T, higher respiratory rate, and increased WOB. Premature breath termination with double-triggering often occurred with a higher flow-termination setting (Fig. 23). Chiumello et al[47] evaluated rise time and flow termination in patients recovering from acute lung injury, and receiving PSV. They found that the fastest rise time reduced the WOB, and the lowest cycling flow reduced the respiratory rate and increased the V_T with no change in the WOB (Fig. 24).

Some new-generation ventilators allow adjustment of the flow at which the ventilator cycles during PSV (see Table 1). Modifications of flow-cycle criteria may need to be carefully adjusted during PSV, and waveforms may assist in adjusting flow termination to a level appropriate for the patient. Using a mathematic model, Yamada and Du[24] showed that the ratio of the flow at the end of patient inspiratory effort to peak inspiratory flow is a function of the patient's respiratory mechanics and the pressure support setting (Table 2). They suggested that the flow-termination criteria during PSV should not be fixed and its setting should be automated so that it varies breath-to-breath, as appropriate, to allow the ventilator to cycle in synchrony with the patient's neural inspiration.[24,48] Tassaux et al validated the model of Yamada and Du in 28 intubated patients undergoing PSV.[49]

Another issue with PSV is the presence of leaks (eg, bronchopleural fistula, cuffless airway, mask-leak with noninvasive ventilation). This may be particularly problematic when providing noninvasive ventilation for patients with obstructive lung disease.[44,45,50] If the leak exceeds the termination flow at which the ventilator cycles, either active exhalation will occur to terminate inspiration or a prolonged inspiratory time will be applied. With a leak, either pressure-controlled ventilation or a ventilator that allows an adjustable flow-termination should be used.

Pressure Support With a Sigh

Sighs in conjunction with PSV may counteract the tendency for lung collapse associated with low V_T and thus improve gas exchange. This was studied by Patroniti et al.[51] They applied sighs in conjunction with pressure support in 13 patients and reported that sighs were associated with P_{aO_2} improvement, an increase in end-expiratory lung volume, an increase in respiratory-

system compliance, and a decrease in respiratory drive. Sighs can be used with PSV on the Puritan-Bennett 840 (bi-level mode) and the Dräger Evita 4 (PCV+ mode). The sigh rate is set at 1–4 breaths/min, the pressure during the sigh is set at 25–30 cm H_2O, and the sigh duration is 2–4 s (Fig. 25).

Summary

PSV has been effectively used to ventilate many patients. However, it has become increasingly appreciated that pressure support may not be a simple mode of ventilation. Issues related to triggering, rise time, and cycling during PSV should be appreciated, and ventilator waveforms may assist with the proper setting of those.

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Discussion

Benditt: Thanks for focusing us back on the patient, which I always think is critical. There's been a lot of effort focused on the ventilator and what it can do, but how it integrates with the patient is the critical thing. It gets back to the BiCore monitor or other methods for looking at the work of breathing. We are still missing the monitor of the patient effort, comfort, and so forth. You could say, why not go to the bedside and just assess it?

Hess: Use the "eyeball test."

Benditt: Right; we'll adjust the machine by looking at the patient. But is there something we could be measuring to try to get to this really bottom line, especially in the weaning process?

Hess: What you're asking for may not be easily attainable with present technology.

Benditt: Maybe what we need is an EMG [electromyogram] of the abdominal muscles or something?

Nilsestuen: There have been a number of articles about using the EMG as the ideal signal to evaluate patient-ventilator synchrony.[1–4] The EMG is the variable most aligned with true neural effort, and is not hindered by the delay times associated with other kinds of transducers. So the EMG is the ultimate signal, and the question is whether we can develop techniques to measure the EMG in a less invasive way that is stable and comfortable for the patient. If such a technique was available, that would definitely be the way to do it.

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Hess: Sinderby et al[1] used a gastric tube with sensors at the diaphragm and used diaphragmatic EMG to trigger the ventilator. Maybe we could use something like that and a surface elec-

trode on the abdomen. You could use that setup to both trigger and cycle the breath, though the cycling part has not been looked at, as far as I know.

REFERENCE

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MacIntyre: Yours is one of the best descriptions I've seen of the cycling problem with pressure support. We recognized this problem several years ago, thanks to the work of Martin Tobin's group.[1] In our institution we don't use pressure support that much anymore, for just that reason. We're really concerned that cycling is an issue; it is difficult to set it right, and these tools you've described are not readily available. We've switched to what we call pressure-assist—a mode that has been around for 20 years. It's the pressure control mode on most ventilators—and if you set the rate to very low or zero, patients can still trigger those breaths. These breaths begin just like a pressure support breath: you set a pressure target; they're patient-triggered; they have all the rise time characteristics you described. The only difference between it and the pressure support breath is that you, the clinician, have control over the inspiratory time. You have to set it.

REFERENCE

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Hess: But you've got to set it right!

MacIntyre: Yes, you've got to set it right. But let me return to what Josh Benditt said, and point out that we've gotten fairly comfortable looking at the way a patient breathes, and looking for those little pressure spikes in the waveform that say that the pressure breath is going too long, or the little sucking that occurs at the end that says a breath is too short. I would submit to you that maybe we've got something simple right now to use, if we're smart enough to recognize and watch the patient's inspiratory and expiratory efforts, that we might be able to set the inspiratory time and use pressure-assist to achieve all the goals and solve the cycle synchrony issue. I'm a little nervous saying that, since I'm sitting next to the guru of patient-ventilator synchrony [Jon Nilsestuen], but I thought I'd stick my neck out and throw that out as a possible option to address the cycling issue.

Hess: Like you, we also use pressure control as an alternative to pressure support in some patients with cycle dyssynchrony. My problem with that is that as clinicians we have to get the inspiratory time setting just right, or we have all the same problems that we have with pressure support. I think that what you say can be done, but as clinicians we have to be able to adjust the inspiratory time to go shorter or longer as necessary. Another way we could do that is to use a ventilator that allows adjusting the flow cycle-off criteria, and adjust that up or down during pressure support as the mechanics change. But what you said we do in practice, so I agree with you, but I still am a little nervous about it at times.

MacIntrye: I think adjusting the flow cycling is even more problematic, because if the patient changes his efforts, that can change a lot. The cycle-off time with the flow cycle would change even more than the inspiratory time.

Hess: Point well made. One of the problems you get into is that if you adjust the rise time, that will adjust the flow at the beginning of ventilation, and then for the same fraction of cycle-off criteria it'll adjust the cycle-off flow.

MacIntyre: But the inspiratory time stays the same. The setting will stay the same if you're using pressure assist.

Hess: Point taken.

Sanborn: In 1999 Magdy Younes's group[1] looked at proportional-assist ventilation. The pressure support part was fascinating. They studied the frequency of breath triggering under 2 conditions: low support pressure and high support pressure. At the lower support pressures there were no pressure (or corresponding flow) perturbations during exhalation. But as the support pressure was raised, both peak inspiratory flow and inspiratory time increased. Then there were pressure and flow perturbations during exhalation. On the assumption that those perturbations reflected failed inspiratory trigger efforts, they plotted the interval between the inspiratory trigger and the following perturbation against the corresponding pressure signals from an esophageal balloon. As I recollect, the correlation was excellent. The study demonstrated that over-support can cause patient-ventilator dyssynchrony. The point was that a lot of clinicians don't pay enough attention to the pressure support level, and they necessarily increase inspiratory time, which disrupts the normal cycle frequency of the patient's respiratory control center, causing the diaphragm to contract during exhalation. And in the end you get patient-ventilator dyssynchrony. But if you reduce the pressure support level, maybe that's coming back to what you're doing—you better promote patient-ventilator harmony.

REFERENCE

1. Giannouli E, Webster K, Roberts D, Younes M. Response of ventilator-dependent patients to different levels of pressure support and proportional assist. Am J Respir Crit Care Med 1999;159(6):1716–1725.

Benditt: I think this is also a big problem in noninvasive ventilation, which we use a lot. Ventilator-patient synchrony is so crucial to the patient's comfort and acceptance of noninvasive ventilation at home. The Quan-

tum noninvasive machine was ahead of its time; 10 years ago it had the rise time setting, which made a tremendous difference in patient acceptance. The BiPAP machine did not have adjustable rise time, and for a lot of our neuromuscular patients it was very difficult to tolerate.

Hess: But the Respironics BiPAP Vision now has the rise time adjustment.

Benditt: Yes. Now almost all of them have adjustable rise time, and it has much improved patient acceptance of these machines at home.

Hess: I agree. Getting back to Neil's point, some of the newer BiPAP machines also allow you to set the maximum inspiratory time, which also improves patient-ventilator synchrony, particularly in the case of leaks.

Pierson:* The term "rise time" has troubled me ever since the first time I saw it. It should be "rise per time" because when we say a faster rise time, we mean faster rate of rise. When we say slower rise time, we mean a more gradual rate of rise. But people are usually using the term in a way that's technically opposite of its intended meaning. More time means slower rise, and faster rise means less time. I don't think the words are used the way they really should be used.

Hess: I take your point, although the way that the terms are sometimes used—the way I think about it—is that rise time is the amount of time that is required to reach the pressure.

Pierson: The term ought to be modified.

Hess: It really has to do with slope; and in fact the way that I modeled it mathematically, it really is slope.

Pierson: It's like trigger sensitivity, which we've always been drawn to. The more sensitive, the less effort it should take, but that's not the way we use it.

Hess: It becomes very confusing because there's no consistency among manufacturers, and making it a bigger number on some machines means it takes *more* time to reach the pressure, and on others it means it takes less. Warren, you were going to add something to that?

Sanborn: In defense of good language, we wrestled with this mightily and came up with "flow acceleration"—that's what it really is—and everybody hated it. They just crucified it, so we went back to rise time.

Hess: That's what it is, and I tried to make the point that the value of that is how it affects the flow, not by how it affects the pressure. But clinicians think about it as the pressure rise, or pressure rise per time.

Nilsestuen: I suppose it depends a little bit on what kind of valve you're talking about, but in some mechanical ventilators it's related to the rate at which the valve opens. So when I give lectures or teach students, I use the rate of valve opening as a way to describe it, because if it opens quickly then the gas flow increases rapidly; if the valve opens slowly, then gas flow increases gradually. That seems to be fairly clean, at least in terms of the concept.

Shrake:* I just want to echo Josh Benditt's comments that at some point we have to look at the patient, and we've got some great tools. You've been in the field long enough that you've ventilated patients without *any* of these tools. When they teach pilots how to fly by instruments, they tell them, "Always trust the instruments; never trust your senses, because your senses will kill you." How frequently do you see a difference between your clinical assessment and what these tools are telling you, and what do you trust, and why?

Hess: I guess I'm going to weasel on this one a little bit and say that we need both. I think that if we have these tools—waveforms and graphics and so forth—and we have an astute clinician at the bedside who can do a *good* patient examination, then I think we have the best of both worlds. In my practice I look a lot at the graphics and the waveforms, but Scott Harris and Luca Bigatello will tell you that I also look at the patient. They've seen me put my stethoscope on the chest.

Sanborn: What did you call that device? Stethoscope?

Hess: In one of my noninvasive ventilation lectures I talk about this. I show a slide that has nice graphics on a noninvasive ventilator, and I say that when I initiate noninvasive ventilation, I look at the graphics but I also still like the good old-fashioned eyeball test, and there's a picture of my eye.

Nilsestuen: Your Figure 17 showed that as they increased the rate at which the valve opened, they reached a point where it was optimal for the patient, and then they went past that, to where it opened so fast that the esophageal pressure or the P_{mus} actually got greater again. Do you have any thoughts about that? I think maybe that's a feedback response from the lung, that it might be irritant receptors or something, but I didn't know if anybody has an explanation for why, if you give it too fast all of a sudden the patient goes into this new zone where

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it's more work rather than less work, which doesn't make sense from the perspective of gas physics.

MacIntyre: That slide, Jon [Nilsestuen], indicates not that they did *more* inspiratory work; what we found is that they would fight against it and the tidal volumes plummeted and we got a lot of expiratory activity. Those waveforms Dean showed are different from what *we* observed. We observed patients actually fighting against the very rapid flow. They didn't like it coming in so fast and they prematurely terminated the breath. So we didn't see what you did.

Bigatello: There is one thing that might help in answering your question. I interpreted it as dyssynchrony. It represents the point at which the patient does not like what he is getting from the ventilator. It doesn't totally explain that, but in that study we also looked at a subjective patient-comfort score, and very high rise time was not the most comfortable. The most comfortable rise time was in the middle. With too slow a rate of rise, patients were not getting enough flow, so you might think that the highest rise time would be more comfortable, but that wasn't true. They were most comfortable in the middle, which is also where they have the least-negative deflection of esophageal pressure. In the last 2 panels are where the patients are *not* comfortable, and somehow they must be dyssynchronous with the ventilator, and that's why they are making their own efforts.

Hess: Let me propose something else. With the increasing pressurization rate—"rise per time" for Dr Pierson—the flow is very high, the ventilator cycles off sooner, and the inspiratory time is shorter. That lowers the tidal volume, so if the patient is going to defend his tidal volume, he has to make a greater inspiratory effort. Feel free to debate!

Bigatello: You have to set the inspiratory time!

Nilsestuen: Dean, I guess I like that explanation a little better; the reason being that if the patient's discomfort causes them to resist the inspiratory flow, their esophageal pressure should go the opposite way. It should become sharply positive to resist the flow, and that's not what this [Figure 17] shows. This shows that the esophageal pressure continues to decline, indicating more inspiratory effort, and that's what seems so counterintuitive.

Hess: That would highlight the point about setting the inspiratory time, rather than having it flow-cycle, because by changing the pressurization rate—the rise time—it changes the flow, and then that changes where the ventilator cycles off if the cycle-off criterion is a fixed percentage of peak flow.

Harris: The only thing about that is that it assumes that somehow the respiratory center *knows* that it's not going to get enough tidal volume, because if you look at the esophageal pressure, it's actually—

Hess: But I don't think these are breaths in sequence.

Bigatello: So those aren't actually matched in time?

Hess: Yes, they are matched in time, but they are not one breath after the other, and because they're *not* one breath after the other, if the tidal volume drops, the P_{CO_2} will go up a bit, and that will increase the respiratory-center output. There will be more respiratory-muscle contraction, esophageal pressure deflection, and tidal volume flow, all in an attempt to lower the P_{CO_2}.

Harris: So it would have to be a physiologic change that has already occurred, and the respiratory center is sensing that and then responding to it.

Hess: Right.

Dhand: One piece of information that would help in that respect is to know what happened to the frequency of breathing in those patients. I think that what Neil is referring to is possible—that when the Hering-Breuer reflex gets activated, that shortens the inspiration. Because inspiration and expiration are linked, then expiration gets shortened too, and that tends to increase the frequency of breathing, and the respiratory drive. When the respiratory drive is increased, you could get a more negative deflection on the esophageal pressure waveform.

Benditt: I want to clarify one point. When you say neural inspiratory time, how do you measure that? What is that?

Hess: That's the time of the respiratory-center output, and it's not easy to measure. Some investigators have spent a lot of time trying to measure that, but essentially what it means is the amount of time that there is an output from the respiratory center.

Dhand: You can measure it if you are looking at the diaphragmatic EMG. The time for the activity of the diaphragm gives you an idea of the neural inspiratory time, and that's really where a lot of the problems with pressure support arise, because one controller is in the patient's brain and then the other controller is in the ventilator, and the two are not matching.