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Pressure Control Ventilation

Dane Nichols, MD*, Sai Haranath, MBBS

Division of Pulmonary & Critical Care Medicine, Oregon Health & Science University, 3181 SW Sam Jackson Park Road, Mailcode UHN-67, Portland, OR 97239, USA

As mechanical ventilators become increasingly sophisticated, clinicians are faced with a variety of ventilatory modes that use volume, pressure, and time in combination to achieve the overall goal of assisted ventilation. Although much has been written about the advantages and disadvantages of these increasingly complex modalities, currently there is no convincing evidence of the superiority of one mode of ventilation over another. It is also important to bear in mind that individual patient characteristics must be considered when adopting a particular mode of ventilatory support. As emphasized in the 1993 American College of Chest Physicians Consensus Conference on Mechanical Ventilation, ''although the quantitative response of a given physiologic variable may be predictable, the qualitative response is highly variable and *patient specific*'' [1].

Partly because of the inherent difficulties in working with pressure ventilation, the Acute Respiratory Distress Syndrome (ARDS) Network chose to use a volume mode of support for their landmark low tidal volumetrial [2]. The preference for volume ventilation at ARDS Network centers was later demonstrated in a retrospective study of clinicians' early approach to mechanical ventilation in acute lung injury/ARDS. Pressure control was used in only 10% of the patient population before study entry. There was a modest tendency to use pressure control ventilation (PCV) in patients with more severe oxygenation defects (PaO_2/FiO_2, or $P/F < 200$) and a greater tolerance for higher airway pressures when using this mode. Volume control ventilation (VCV) in an assist-control or synchronized intermittent mandatory mode, however, was clearly a preferred method of support [3].

PCV may offer particular advantages in certain circumstances in which variable flow rates are preferred or when pressure and volume limitation is required. These desirable characteristics of PCV, however, can produce

* Corresponding author.
E-mail address: nicholda@ohsu.edu (D. Nichols).

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unanticipated consequences when ventilatory strategies used in volume modes are similarly applied in pressure-regulated ventilation. The goal of the following sections is to provide clinicians with a fundamental understanding of the dependent and independent variables active in PCV and describe features of the mode that may contribute to improved gas exchange and patient-ventilator synchronization.

It is important to stress that any method of mechanical ventilation may contribute to secondary forms of injury in heterogeneous lung disease and that the injury incurred is currently beyond our capability of recognizing at the bedside. Developments in our understanding of pressure-volume curves and the recent demonstration of microscopic shear and stress injury in animal models of ventilator-induced lung injury call into question the whole concept of "safe" ranges of pressure and volume in mechanical ventilation [4–8]. As we explore the characteristics of flow and pressure generation in PCV, we draw attention to those aspects of pressure ventilation shown to be associated with adverse outcomes in experimental settings. In this way we hope to provide clinicians with a balanced framework in which to choose the most appropriate method of ventilatory support.

Physiology of pressure control ventilation

PCV, unlike volume targeted modes, is pressure and time cycled and generates tidal volumes that vary with the impedance of the respiratory system. A working understanding of the factors that determine volume delivery is necessary for proper implementation of this mode of ventilation. During the inspiratory phase of PCV, gas flows briskly into the ventilator circuit to pressurize the system to a specified target. Once the target pressure has been reached, flow is adjusted to maintain a flat or "square wave" pressure profile over the remainder of the set inspiratory time [9,10]. This goal is achieved by sampling airway pressure approximately every 2 msec to provide critical feedback to flow controller mechanisms within the ventilator. By tracking the rate of change in pressure during inspiration, appropriate deceleration can occur as the pressure ceiling is approached. If the gradient between the circuit pressure and pressure target is large, flow is brisk. As the gradient between the recorded pressure and preset target narrows, flow decelerates to prevent overshoot. When impedance to flow is modest, the resulting flow curve demonstrates uni-exponential decay [11]. In situations of airflow obstruction, pressure targets are typically reached at lower flow rates, which contributes to a decelerating ramp profile (Figs. 1 and 2).

Flow into the ventilator circuit continues until conditions relating to pressure and time are met. Once the pressure within the alveolus rises to the level of the ventilator circuit, the gradient driving flow no longer exists and flow ceases. This process has important implications for tidal volume delivery in situations of altered compliance and resistance, as discussed later. In the PC mode of ventilation, the inspiratory time (I time) over which the pressure

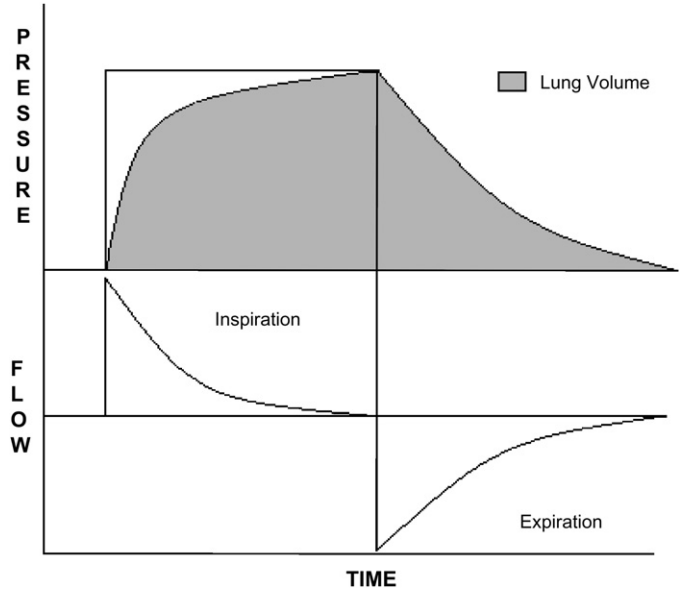

Fig. 1. Idealized pressure and flow time curve in PCV. A "square wave" pressure profile is achieved in inspiration by rapidly delivering flow into the ventilator circuit. Once the pressure target is met, flow rates decay in a uni-exponential manner, reaching a no-flow state at the termination of the inspiratory cycle. The bulk of the tidal volume is delivered early in inspiration when flow rates are maximum.

profile is maintained must be specified by the clinician. Shortening the inspiratory fraction of the duty cycle can lead to cessation of flow well before equilibration of circuit and alveolar pressures occurs. Extension of I time beyond the point of equilibration, on the other hand, increases mean airway pressure but generally does not lead to a further increase in tidal volume. Fortunately, the inspiratory time necessary to achieve pressure equalization can be established easily through various simple bedside maneuvers (see later discussion). Finally, to avoid potentially harmful pressure increases, flow ceases when circuit pressure exceeds the preset target by approximately 3 cm H_2O. This safeguard presumably decreases the risk of barotrauma during episodes of forced expiration, gas trapping, and vigorous coughing.

As a result of the high initial flow rates and large circuit-to-alveolus pressure gradient, a large percentage of the tidal volume is delivered early in the inspiratory cycle. During the ensuing pressure buildup, flow rapidly decelerates, which translates into a small volume of transported gas at the end of the inspiratory phase. Because of the disproportionate weighting of tidal volume delivery in early inspiration, the mean airway and alveolar pressures tend be higher in PCV compared with VCV when a constant flow rate (square wave) is used. The decelerating flow profiles characteristic of PCV, however, can be easily mimicked in volume modes of ventilation by adopting similar decelerating flow profiles. By using ramp waveforms and

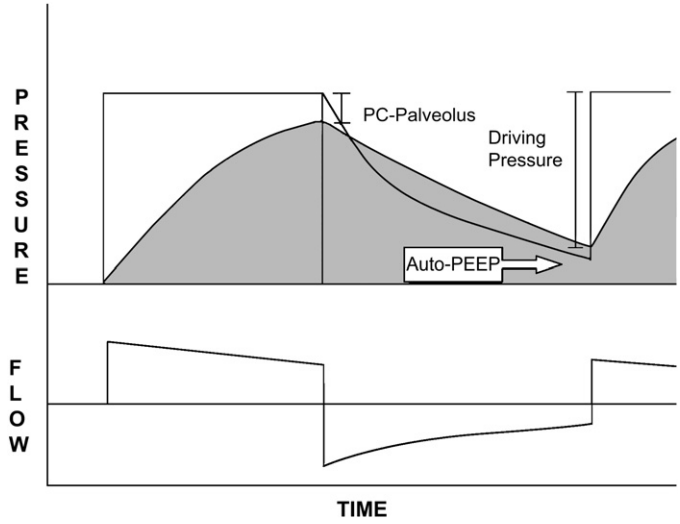

Fig. 2. Pressure and flow curves in obstructive disease states. The initial flow rates needed to reach the pressure target are reduced. Flow continues throughout the inspiratory cycle because of the slow equilibration of circuit and alveolar pressures. This process leads to ''squaring up'' of the flow profile. The slow delivery of gas and the decrease in pressure gradients driving flow lead to a reduction in tidal volume.

inspiratory holds in VCV, pressure and flow curves and parameters of gas exchange are similar in stable patients [12].

Determinants of tidal volume and minute ventilation

The tidal volume output that results from a given applied pressure and inspiratory time is predominantly influenced by flow resistance and respiratory system compliance [13]. If resistance to flow is high, the flow component of impedance is dissipated slowly over resistive elements, which results in small driving pressures across the circuit. Under these circumstances, pressure targets are reached at lower initial flow rates and must be maintained for longer periods of time to ensure equilibration with alveolar lung units. Research has shown that the flow curves assume a shallower slope and that the decay profile becomes more linear as obstruction increases [14]. As the rate of flow decreases with increasing resistance, tidal volume may fall if no adjustment is made in inspiratory time.

Simple observations at the bedside can assist the clinician in determining whether I time is adequate to achieve equilibration of circuit and alveolar pressures under conditions of airflow resistance. If a steady state has not been achieved at the end of the inspiratory period, an inspiratory hold maneuver is associated with a fall in airway pressure below the pressure control target. In the absence of significant gas trapping, prolonging inspiratory time then leads to an increase in delivered tidal volume. If graphic displays

are available for review, flow-time curves demonstrate continuing gas delivery throughout the inspiratory cycle without a period of zero flow being evident on inspection. Modifying the duty cycle to the point at which inspiratory flow has ceased should produce the largest tidal volume for a given static compliance provided it does not lead to an increase in auto-positive end-expiratory pressure (PEEP) (Figs. 3 and 4).

Low tidal volumes that stem from alterations in respiratory system compliance on the other hand are influenced to a lesser extent by adjustments in inspiratory time. In the absence of airflow limitation, flow rates are brisk and lead to rapid equilibration of circuit and alveolar pressure (Fig. 5). The delivered tidal volume then depends primarily on the pressure applied over the duty cycle and the static compliance of the respiratory system as predicted by the following equation:

$$C_{ST} = V_T/[P_{Plat} - PEEP\ (\text{or auto} - PEEP)]$$

$$V_T = \Delta P \times C_{ST}$$

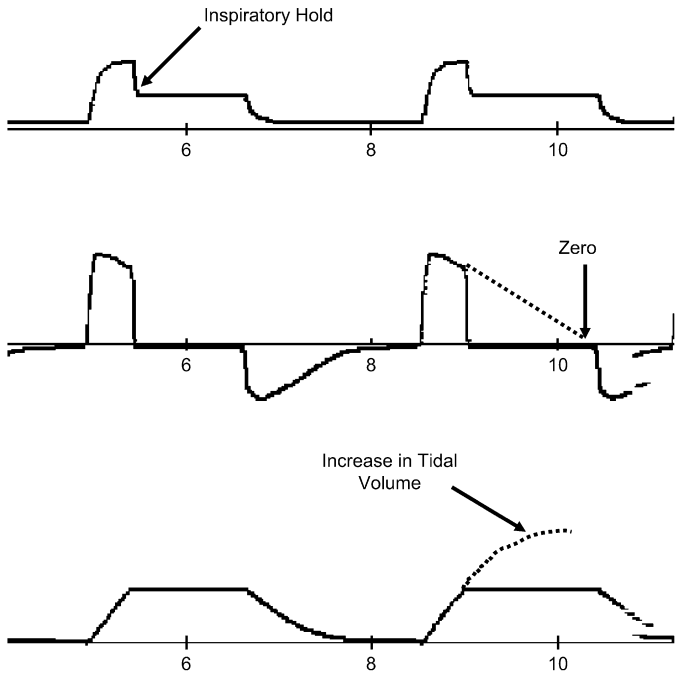

Fig. 3. PCV with short inspiratory times. The flow–time curve demonstrates abrupt cessation of flow well above the zero-flow point. Repetitive inspiratory hold maneuvers reveal large pressure drops that result from small delivered tidal volumes. In this instance, prolonging inspiratory time will lead to an increase in delivered tidal volume. (X-axis:Time.Y-axis:Top panel:Pressure; Middle panel:Flow;Bottom panel:Tidal Volume.)

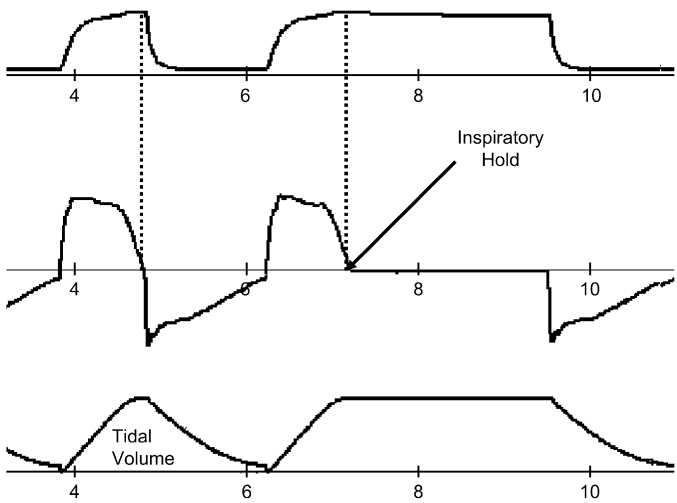

Fig. 4. In the initial breath, flow ceases at the end of the inspiratory cycle. During the subsequent breath, an inspiratory hold maneuver yields a stable pressure curve. These observations are consistent with pressure equilibration between the test lung and the ventilator circuit at the end of the regular inspiratory cycle. Prolonging inspiratory time in this circumstance would fail to increase tidal volume. (X-axis:Time.Y-axis:Top panel:Pressure;Middle panel:Flow;Bottom panel:Tidal Volume.)

in which C_{ST} represents static compliance, V_T represents tidal volume, P_{plat} represents plateau pressure, and *PEEP* represents positive end-expiratory pressure. As the respiratory system compliance decreases, tidal volume falls and vice versa. It is particularly important to recognize the impact of changing compliance on delivered tidal volume during maneuvers such as proning or in circumstances of high intra-abdominal or intrathoracic (eg, pneumothorax) pressure. Cycling frequency also has been shown to influence tidal volume delivery. At high respiratory rates, the ability of ventilators to create rectilinear pressure profiles deteriorates. Recent evidence suggests that this is particularly true of certain models of mechanical ventilators used in the anesthesia theater [15]. As the ability to seek the pressure target declines, delivered tidal volume may be reduced. This feature of PCV, along with other determinants of tidal volume, makes it difficult to predict the impact of frequency changes on overall minute ventilation.

This seems particularly true in situations of airflow resistance. At high cycling frequencies, gas trapping can result from expiratory flow limitation. The resulting increase in mean alveolar pressure then limits flow into the system, which leads to a drop in delivered tidal volume. The relationship between mean airway pressure and mean alveolar pressure has been formalized in the following equation [16]:

$$\text{mean } P_{Alv} = \text{mean } P_{AW} + (V_E/60)(R_E - R_I)$$

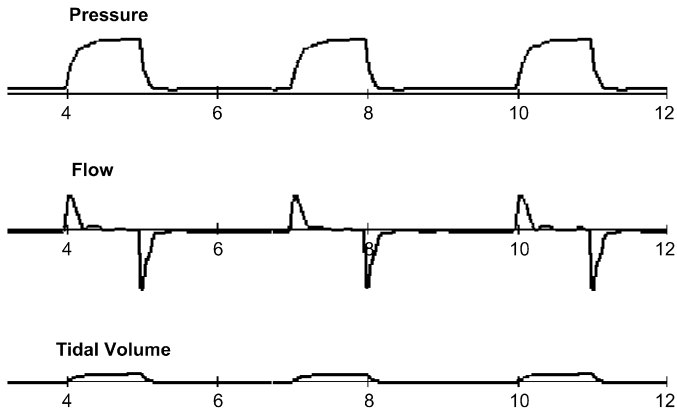

Fig. 5. A simulated PCV breath under conditions of decreased static compliance. Initial flow rates are high and quickly decelerate once the pressure target is achieved. Delivered tidal volumes are markedly reduced. (X-axis:Time.Y-axis:Top panel:Pressure;Middle panel:Flow; Bottom panel:Tidal Volume.)

in which P_{Alv} represents the average alveolar pressure over a respiratory cycle, P_{AW} represents the average circuit pressure over the respiratory cycle, V_E represents minute ventilation, and R_E and R_I represent expiratory and inspiratory resistance, respectively. From inspection of the equation, it is apparent that in the setting of high minute ventilation and increasing airflow obstruction, dynamic recordings in the external circuit will underestimate alveolar pressures. At the bedside, unappreciated gas retention can result in falling tidal volume and minute ventilation with resultant hypercapnia. Increasing respiratory rate under these circumstances in an attempt to improve minute ventilation generally is met with further deterioration in gas exchange parameters (Fig. 6).

Mathematical modeling of PCV has led to a more thorough understanding of the determinants of minute ventilation under conditions of increased resistance [14,17]. As respiratory rates increase, minute ventilation rises to a point at which inspiratory and expiratory airflow limitation create a unique boundary that caps any further increase in minute ventilation. At extreme rates, gas delivery begins to fall as driving pressures are reduced by the development of auto-PEEP and limited available time for inspiration. In dealing with patients with obstructive lung disease, clinicians must be aware that the expected increase in minute ventilation that results from changing respiratory rates quickly reaches a theoretic maximum determined by the degree of airflow obstruction.

Increasing respiratory rates at the limits of minute ventilation also can lead to worsening gas exchange by altering dead space to tidal volume ratios (V_D/V_T). Once minute ventilation approaches the bounding limit, tidal volumes begin to fall. As a percentage of the breath, dead space ventilation

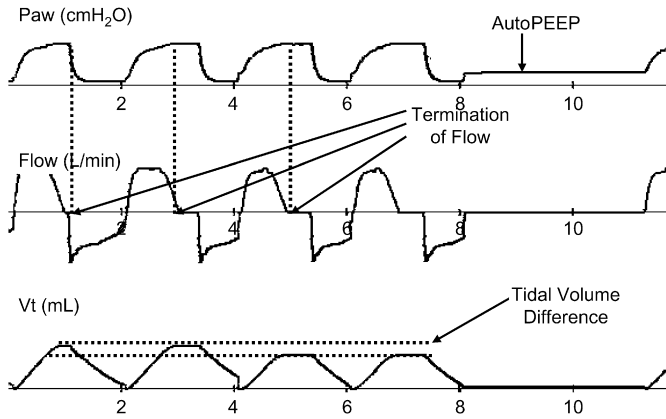

Fig. 6. The rapid development of auto-PEEP resulting from long inspiratory times and high respiratory rates leads to early cessation of flow and reduced tidal volumes. Auto-PEEP is demonstrated during expiratory hold maneuver. (X-axis:Time.Y-axis:Top panel:Pressure;Middle panel:Flow;Bottom panel:Tidal Volume.)

then increases, which leads to less effective CO_2 clearance. Animal models of PCV have demonstrated the development of progressive hypercapnia over relatively stable minute ventilation as cycling frequencies were increased [18]. Calculated dead space ratios revealed a strong tendency to change in the direction of the respiratory rate [18].

The response to changes in respiratory rate is different when impedance is dominated by the elastance of the respiratory system. In these circumstances, minute ventilation generally increases along with cycling frequency [11,17]. The slope of this relationship is determined by the individual static compliance. If the respiratory system is stiff, delivered tidal volumes are small and result in modest increases in minute ventilation. In the absence of significant gas trapping, the impact of cycling frequencies on dead space ratios in restrictive lung disease would be expected to be considerably less than seen in obstructed states.

Theoretic advantages/disadvantages of pressure control ventilation

Interest in PCV and decelerating waveforms dates back several decades. Following the description of ARDS in the late 1960s, numerous animal studies were published that investigated the relative contribution of tidal volume, peak airway pressure, and end-expiratory pressure to ventilator-induced lung injury. These investigations, along with observational studies, suggested that high peak airway pressures were associated with macro- and microscopic barotraumas [8,19–21]. PCV, by virtue of its pressure ceiling, was seen as a possible means of avoiding transient high peak alveolar pressures in lung units close to central airways and possessing fast time constants. In conditions of heterogeneous lung pathology, significant

differences in regional pressure and volume may arise from local conditions that influence flow and compliance [6,7]. Pressure limits and decelerating flow profiles are thought to produce more uniform distribution of forces within the lung, possibly reducing the risk of volu- and barotrauma.

The decelerating waveform also may produce other advantages. Because the bulk of the tidal volume is delivered early in the respiratory cycle, the mean airway pressure over the duty cycle is increased [13]. Modell and Cheney [22] demonstrated that decelerating waveforms improve oxygenation in the setting of diffuse lung injury when compared with accelerating and square wave patterns of tidal volume delivery. Both of the latter profiles result in lower mean airway pressures and presumably produce less recruitment of poorly ventilated lung units. As might be expected from the effect on mean airway pressure, choice of flow pattern also can lead to improved lung mechanics. In a comparative trial of flow patterns in 14 patients with respiratory failure, Al-Saady and Bennett [23] demonstrated higher static and dynamic compliance along with improved measures of work of breathing when decelerating flow profiles were used. Changes in compliance that result from different flow profiles depend primarily on the percentage and threshold opening pressure of atelectatic lung units [4]. Any improvement in these measures likely will occur early in the course of lung injury and are affected by the nature of the insult [24]. A final mechanism by which flow profiles can influence the inflationary properties of the respiratory system involves changes in the "nonlinear" or viscoelastic behavior of the lung. Viscoelastance is responsible for the pressure drop that occurs between the time that flow ceases in the airways and a stable plateau pressure is achieved. The decay in pressure reflects time constant inequalities and tissue viscance and has been associated with the degree of lung injury. When various modes of ventilation were randomly applied in acutely ill patients, Edibam and colleagues [25] demonstrated that PCV was associated with the smallest viscoelastic pressure drop when compared with VC and pressure control inverse ratio ventilation modes. The clinical implications of these findings, however, remain unknown.

PCV also may significantly lower work of breathing in patients with variable or high drives to breathe [26,27]. In VCV, flow rates are fixed and generally determined by the respiratory therapist. If a patient's demand for flow exceeds the set rate, the patient may continue vigorously inspiring in response to internal cues. The sustained, high negative intrathoracic pressure contributes to additional work of breathing. Inappropriate flow rates are easily identified by examining pressure-time curves. If flow is inadequate, the inspiratory arm of the curve has a "scooped out" concave appearance (Fig. 7) [28]. Adjusting flow rates or changing to a pressure-regulated mode of ventilation often improves comfort and apparent respiratory effort. When flow rates are variable and determined by pressure targets, as in PCV, changing patient demand is met by similar directional changes in delivered flow. These changes avoid "flow starvation" commonly encountered in volume forms of ventilation (Fig. 8).

Specifying pressure and inspiratory time also may have implications for gas exchange and minute ventilation by additional mechanisms. Puddy and Younes [29] have demonstrated that respiratory frequency can be influenced by altering flow rates in normal individuals leading to ''flow related tachypnea'' [30]. By shortening inspiratory time through use of high flow rates, respiratory frequency increased in study subjects by approximately 60%. The response was thought to be mediated through the Hering-Breuer reflex that influences neural inspiratory and expiratory cycling. Decelerating patterns of flow, in contrast to square wave profiles, produce longer I times for a given tidal volume and may encroach on neural expiratory time, leading to changes in breathing frequency. This neuromechanical coupling may decrease the risk of flow-related hypocapnia that results from shortened inspiratory times.

Not all studies have shown benefits from pressure-regulated flow, however. Dembinski and colleagues [31] demonstrated that once PEEP was optimized, a square wave flow pattern produced a more favorable V/Q distribution than decelerating ramps in an animal model of acute lung injury. It seems that any improvement in gas exchange that arises from an increase in mean airway pressure (mAWP) depends on the amount of lung tissue available for recruitment. In the absence of recruitable, atelectatic lung units, increasing airway pressure leads to decreased cardiac output and, ultimately, worse gas exchange. A decelerating pattern also may conceivably lead to overinflation in regions of the lung with relatively normal

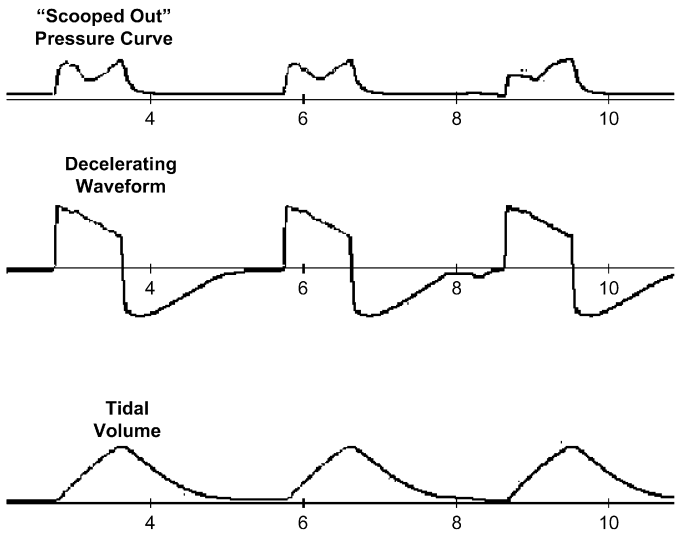

Fig. 7. Volume control breath with a decelerating ramp waveform. An exaggerated inspiratory effort is simulated midway through the inspiratory phase. The fixed flow pattern fails to meet the new flow demand, which leads to a ''scooped out'' appearance in the pressure time curve. (X-axis:Time.Y-axis:Top panel:Pressure;Middle panel:Flow;Bottom panel:Tidal Volume.)

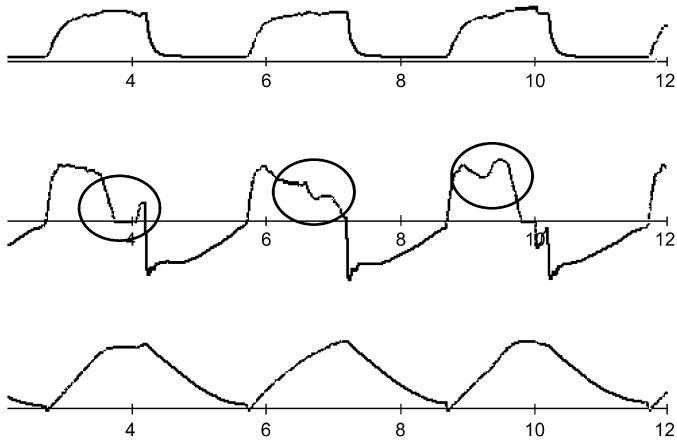

Fig. 8. Pressure control breath demonstrates variable flow. Initial breath: simulated early exhalation. Middle breath: simulated transient midcycle resistance. Final breath: simulated midcycle increased flow demand. (X-axis:Time.Y-axis:Top panel:Pressure;Middle panel:Flow;Bottom panel:Tidal Volume.)

mechanics. By comparing inspiratory and expiratory CT scanning in an ovine lung injury model, Roth and colleagues [32] noted a modest increase in high attenuation (ie, overinflated) lung regions. This small difference in density distribution was not reflected in either lung mechanics or gas exchange.

There are additional concerns regarding the use of PCV as a lung protective strategy. Early in the inspiratory cycle flow, rates are at their maximum and produce rapid changes in pressure within the conductance system, which can lead to high shearing forces in distal airways of the lung. In experimental models of ventilator-induced lung injury, high initial flow rates led to greater deterioration in gas exchange, higher dry-wet lung weights, and more severe histiologic evidence of barotrauma when compared with slower rates despite similar peak and plateau pressures [33,34]. Animal models of ventilator-induced lung injury also suggest that use of PCV with long inspiratory times leads to greater lung injury [35,36]. Several mechanisms have been postulated, including prolonged exposure to alveolar wall stress and greater opportunity for uniform equilibration of alveolar pressures with ventilator circuit pressures. It is clear that creating lower peak airway pressures through decelerating flow profiles does not provide protection from secondary forms of ventilator-associated lung injury, although this approach to ventilatory support is commonly undertaken in the belief that it occurs. Unfortunately, the relative contributions of shear and stress to secondary forms of injury cannot be judged at the bedside. Awareness of the potential contribution of these forces to ventilator-associated lung injury should temper clinicians' enthusiasm for PCV in situations of heterogeneous lung injury, however.

Use of pressure control ventilation in various disease states

PCV has long been used in the setting of difficult-to-manage acute lung injury/ARDS. Clinicians have exploited the variable flow rates to improve patient work of breathing and limit high peak airway pressures. PCV also generally is associated with increased mean airway pressure, a ventilatory parameter found to correlate with oxygenation status. By delivering a larger proportion of the tidal volume early in the inspiratory phase, the lung is maintained at a higher volume, presumably recruiting alveolar lung units to participate in gas exchange.

Unfortunately, few human trials directly comparing conventional PCV with VCV in ARDS have been performed [37–41]. One of the better known studies was reported by Esteban and colleagues [42] writing for *The Spanish Lung Failure Collaborative Group*. Patients with the diagnosis of ARDS were randomly assigned to either VCV or PCV with adjustment in ventilator parameters to maintain plateau pressures $\leq$35 cm H_2O. Delivered tidal volumes, measures of gas exchange, and estimation of lung compliance were not significantly different over the course of the study. In-house mortality and multi-organ dysfunction occurred more frequently in the VCV arm of the trial but were attributed to differences in baseline characteristics and preceding organ failure. Multivariate analysis suggested that the mode of ventilation did not influence outcome.

With rare exceptions, PCV does not seem to offer any substantial advantage over volume control ventilation in terms of gas exchange or lung mechanics. This finding is particularly true when decelerating ramp waveforms and inspiratory hold maneuvers are used in VCV to mimic the flow profile achieved in PCV [12].

A modification of PCV termed ''pressure control inverse ratio ventilation'' proved to be a popular approach in the late 1980s and early 1990s to the management of patients who have ARDS and refractory gas exchange defects. It is occasionally still used. In this mode, inspiratory time is intentionally extended beyond an I:E ratio of 1:1. Prolonging inspiratory time and limiting expiratory time was thought to lead to two potential benefits: (1) higher mean airway pressure and (2) creation of intrinsic PEEP. Despite pressure control inverse ratio ventilation's theoretic advantages, multiple studies failed to demonstrate any significant improvement in oxygenation status when similar mean airway pressures were generated by matching extrinsic PEEP to the level of recorded intrinsic PEEP [43–45]. In studies that reported improved oxygenation with inverse ratio ventilation, an increase in mean airway pressure generally occurred and was not controlled for [46–49]. Cardiac indices and oxygen delivery often deteriorate at the higher mean airway pressures generated in pressure control inverse ratio ventilation [50,51]. Of note, $PaCO_2$ is either unaffected or modestly improved despite a reduction in minute ventilation. When calculated, the improvement in dead space ratios is small and of doubtful clinical

significance. After review of the available literature, Shanholtz and Brower [52] came to the conclusion that "IRV remains unproven in the management of ARDS."

The use of PCV in obstructive lung disease is even less clear. Few studies have been undertaken in this population, leaving clinicians appropriately skeptical of this mode of ventilation for patients with airway disease. The concern surrounding PCV relates to the impact that inspiratory resistance and auto-PEEP have on delivered tidal volume. Flow rates and circuit-to-alveolar pressure gradients may fall as inspiratory and expiratory resistance increases. Delivered tidal volume may become unpredictable and lead to unintended hypoventilation. In the setting of status asthmaticus, in which airflow obstruction can improve dramatically over the course of the illness, tidal volumes also may increase unexpectedly. Fortunately, modern ventilators track various parameters and allow early detection of changing lung mechanics. By establishing tight alarm thresholds, clinicians can respond quickly to falling or rising tidal volumes and minute ventilation.

The concern over possible hypoventilation and resultant hypercapnia in PCV may be overstated. Tolerance for respiratory acidosis is remarkable, with well-documented case reports describing patients with $PaCO_2$ levels exceeding 150 mm Hg [53,54]. Although systemic vasodilation is a known consequence of profound hypercapnia, hemodynamic parameters are generally well maintained in the absence of significant underlying heart disease or central sympatholysis.

In some ways, pressure control forms of ventilation may represent a safer alternative to VCV in severe airflow obstruction. Although the risk of hypoventilation may increase, the risk of overt barotrauma (eg, pneumothorax, pneumomediastinum) may be diminished. In VCV, as gas trapping develops, pressure can build rapidly beyond the alveolar lung unit's elastic limit. Plateau pressures have been used as a surrogate for alveolar volu- and barotrauma risk in volume modes of ventilation, but these measures do not predict the occurrence of barotrauma and directly measured gas volume at end inspiration. When trapped gas volume exceeds 20/mL/kg, the risk of barotrauma is significant [55,56].

Few centers in the United States use trapped gas volume to guide therapy in status asthmaticus. In the absence of regular monitoring of this parameter, PCV may provide a safer method of ventilating patients. As auto-PEEP increases, driving pressure falls, which results in a smaller delivered tidal volume. "Squaring up" of the flow profile as a result of airflow resistance also may serve to protect lung units with fast time constants from transient high pressures.

Despite these potential advantages, pressure-regulated modes of ventilation have been rarely studied in the management of status asthmaticus. Sarniak and colleagues [57] have reported the largest PCV experience in status asthmaticus to date. In their pediatric population, PCV seemed to be safe and associated with improved gas exchange. A review of ventilatory

parameters revealed average I times of 1 second, I:E ratios of 1:4, and peak inspiratory pressures of 36 to 40 cm H_2O. The duration of mechanical ventilation averaged 29 hours, with only one pneumothorax being recorded. Earlier studies of invasive and noninvasive pressure support ventilation also noted that pressure-regulated breaths can be used effectively in severe airflow obstruction [58–60]. Recent reviews of status asthmaticus suggest that the mode of ventilation is not terribly important as long as gas trapping is avoided. Some authors endorse PCV as a starting mode of mechanical ventilation in patients who have status asthmaticus [61–63].

In patients who have chronic obstructive pulmonary disease, decelerating waveforms are associated with improved dead space ventilation, lower $PaCO_2$, and less dyspnea when compared with either square or sine wave flow patterns. Work of breathing also may be reduced by decelerating flow profiles, particularly if the flow rate is variable and tied to airway pressure targets. Occasionally, clinicians may encounter patients with severe emphysema who possess little elastic recoil in the lung parenchyma. Pressure ventilation under these circumstances can lead to large tidal volumes and subsequent hyperinflation. A restrictive volume mode of ventilation may be preferable in these instances.

Summary

Despite its popularity, PCV has not been proved superior to other modes of mechanical ventilation. Although it is associated with lower peak airway pressures, the impact on lung mechanics, gas exchange, and risk of macro- and microscopic barotrauma is variable.

The adjustable flow rates and pressure limitations may prove useful in certain populations. Patients with high drive to breathe may enjoy a decreased work of breathing with PCV compared with VCV. In patients who have obstructive lung disease, pressure limitation also may diminish the risk of barotrauma and increase the likelihood of unintentional hypoventilation. The role of PCV in other conditions, such as acute lung injury, remains to be defined. The potential for lung recruitment through increased mean airway pressure continues to make this an attractive mode in patients with large shunt fractions. Any potential benefit depends on the nature and timing of the lung injury, however, and may be offset by shear- and stress-related volu- and atelectrauma.

Implementation of PCV requires a practical understanding of the relationship between flow, time, and pressure. Unlike VCV, in which tidal volume is guaranteed, gas delivery in PCV varies in complex ways. The simplistic approach of ''turning up'' the ventilator may lead to unexpected clinical deterioration when using this mode. Careful, repeated observation, however, can make this a safe and effective method of ventilatory support.

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