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Airway Pressure Release Ventilation: Theory and Practice

P. Milo Frawley, RN, MS, and Nader M. Habashi, MD†*

Airway pressure release ventilation (APRV) is a relatively new mode of ventilation, that only became commercially available in the United States in the mid-1990s. Airway pressure release ventilation produces tidal ventilation using a method that differs from any other mode. It uses a release of airway pressure from an elevated baseline to simulate expiration. The elevated baseline facilitates oxygenation, and the timed releases aid in carbon dioxide removal.

Advantages of APRV include lower airway pressures, lower minute ventilation, minimal adverse effects on cardio-circulatory function, ability to spontaneously breathe throughout the entire ventilatory cycle, decreased sedation use, and near elimination of neuromuscular blockade. Airway pressure release ventilation is consistent with lung protection strategies that strive to limit lung injury associated with mechanical ventilation. Future research will probably support the use of APRV as the primary mode of choice for patients with acute lung injury. (KEYWORDS: acute lung injury, airway pressure release ventilation, alveolar recruitment, alveolar derecruitment, lung protective strategies)

Airway pressure release ventilation (APRV) is a mode of ventilation that was first described in 1987.[1,2] It uses a philosophy that differs fundamentally from that of conventional ventilation. Whereas conventional modes of ventilation begin the ventilatory cycle at a baseline pressure and elevate airway pressure to accomplish tidal ventilation (Figure 1), APRV commences at an elevated baseline pressure (similar to a plateau pressure) and follows with a deflation to accomplish tidal ventilation (Figure 2). In addition, during APRV, spontaneous breathing may occur at either the plateau pressure or deflation pressure levels. This article provides a detailed examination of the terminology, indications, theoretical benefits, advantages, and disadvantages of APRV as well as a discussion of application and weaning procedures.

Airway Pressure Release Ventilation Defined

Airway pressure release ventilation has been described as continuous positive airway pressure (CPAP) with regular, brief, intermittent releases in airway pressure.[3,4] The release phase results in alveolar ventilation and removal of carbon dioxide (CO_2). Airway pressure release ventilation, unlike CPAP, facilitates both oxygenation and CO_2 clearance and originally was described as an improved method of ventilatory support in the presence of acute lung injury (ALI) and inadequate CO_2 ventilation.[2,5] Airway pressure release ventilation is capable of either augmenting alveolar

From *Maryland ExpressCare and †Department of Critical Care Medicine, University of Maryland Medical Center, Baltimore, Maryland.

Reprint requests to P. Milo Frawley, RN, MS, Maryland ExpressCare, TGR25C, University of Maryland Medical Center, 22 South Greene Street, Baltimore, MD 21201.

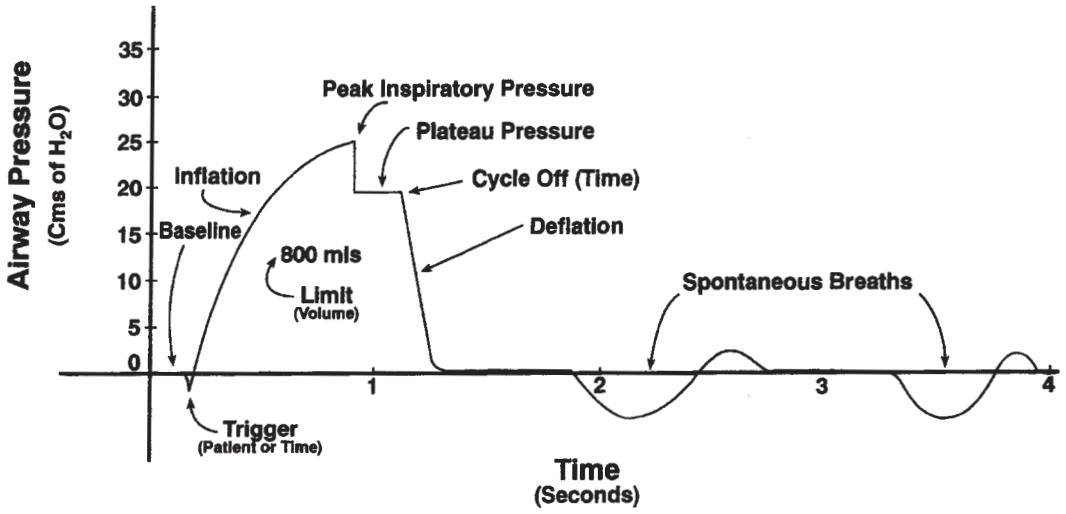

Figure 1. *Conventional volume targeted ventilation, e.g., synchronized intermittent mandatory ventilation (SIMV). Any mechanically delivered breath will be defined by its trigger, limit, and cycle off feature. In SIMV, the breath will be triggered by either the patient or by time, the volume delivered will limit the breath, and time will cycle the breath off into exhalation. Cms of H_2O = centimeters of water.*

ventilation in the spontaneously breathing patient or accomplishing complete ventilation in the apneic patient.[6] The CPAP level drives oxygenation, while the timed releases aid in CO_2 clearance.

Technically, APRV is a time-triggered, pressure-limited, time-cycled mode of mechanical ventilation. In addition, APRV allows unrestricted, spontaneous breathing throughout the entire ventilatory cycle (Table 1). Advantages of APRV include: significantly lower peak/plateau airway pressures for a given tidal volume; the ability to superimpose spontaneous breathing throughout the ventilatory cycle; decreased sedation; and near elimination of neuromuscular blockade use.[7,8] Features that distinguish APRV from other modes of mechanical ventilation include sponta-

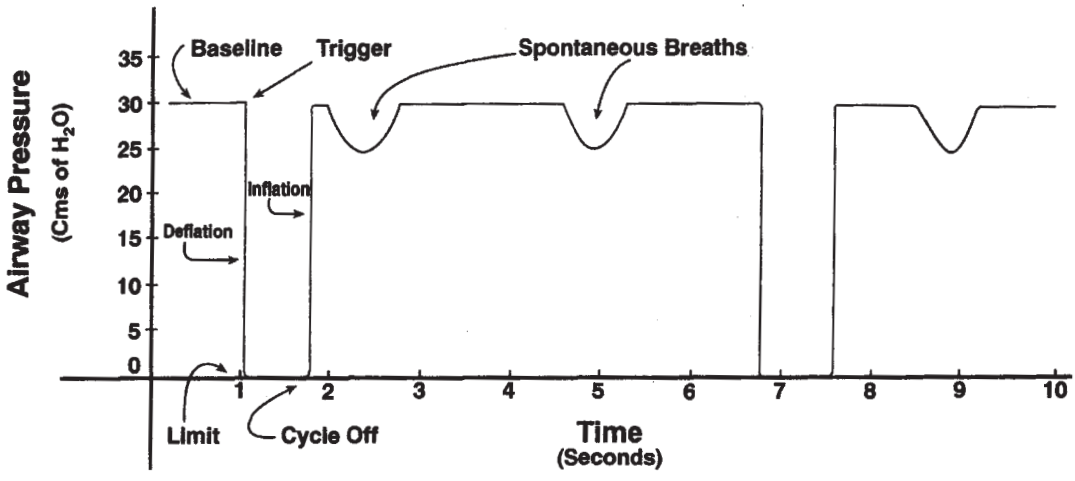

Figure 2. *Airway pressure release ventilation: this can also be defined by a trigger, limit, and cycle off feature. However, unlike other modes of ventilation, the trigger (time) initiates a drop in airway pressure. The amount of pressure change will be the limit. The cycle off will occur because of time. Airway pressure then returns to the baseline. Cms of H_2O = centimeters of water.*

TABLE 1 ■ Classification of Common Modes of Mechanical Ventilation

Mode	Trigger	Limit	Cycle Off	Spontaneous Breathing	Flow of Gas
A-C (volume)	Time or patient	Volume	Time	No	Constant
A-C (pressure)	Time or patient	Pressure	Time	No	Decelerating
SIMV (volume)	Time or patient	Volume	Time	Yes	Constant
PSV	Patient	Pressure	Flow of gas	No	Decelerating
PRVC	Time or patient*	Volume	Time	No	Decelerating
APRV	Time	Pressure	Time	Yes	Decelerating

Note: A mechanically delivered breath is made up of three distinct phases. The Trigger initiates the breath; the Limit will stop the breath from increasing, but does not initiate exhalation; and the Cycle Off, that switches the breath from inspiration to exhalation. Beyond this, modes may or may not allow unsupported, spontaneous breathing. The inspired gas may be delivered using either a constant or decelerating flow of gas.

*This mode is designed for patients with no breathing capacity, though they are able to trigger breaths.

A-C = assist control; SIMV = synchronized intermittent mandatory ventilation; PSV = pressure support ventilation; PRVC = pressure regulated volume control; APRV = airway pressure release ventilation.

neous breathing throughout the ventilatory cycle and an intermittent pressure release phase that results in a brief decrease in lung volume to assist ventilation.[1,2]

☐History of Mechanical Ventilation

The basic principles of ventilator design and management were founded upon patients who developed nonparenchymal respiratory failure (e.g., polio). In the absence of adequate research, those same principles were applied to patients with parenchymal respiratory failure as well (e.g., ALI). Mode selection often was based on availability and simplicity of the ventilator, user experience, and tradition, because little evidence existed to guide management.

In 1993, the American College of Chest Physicians (ACCP) consensus conference failed to "agree on an optimum mode of ventilation for any disease state or an optimum method of weaning patients from mechanical ventilation."[9(p1834)] The ACCP agreed that well-controlled clinical trials that defined the indications and uses of specific modes of ventilation were lacking. New technology must scientifically show a distinct advantage in safety, expense, ease of operation, or therapeutic outcome.[10,11]

Despite more than 30 years since its recognition, acute respiratory distress syndrome (ARDS) continues to have a 30% to 50% mortality rate.[12,13] Recently, discovery of the potential for mechanical ventilation to produce ventilator-associated lung injury has resulted in the development of new lung protective strategies.[14] Lung protective strategies include those described in the "the open lung approach" promoted by Amato et al.[15] The open lung approach uses reduced tidal volumes (6 mL/kg) to prevent high-volume lung injury and over-distension of airspaces. In addition, Amato et al.[16] used elevated end expiratory pressure (average positive end-expiratory pressure [PEEP] 16 cm water pressure), to prevent low volume lung injury from cyclic airway reopening.

The recently completed ARDSNet study compared conventional tidal volume (12 mL/kg) to reduced tidal volume (6 mL/kg).[13] The results of the ARDSNet trial[13] and a study conducted by Amato et al.[16] suggest an association between reduced tidal volume and improved outcome. Although the ARDSNet trial targeted similar PEEP levels in both its groups, study protocols for maintaining saturation resulted in higher levels of set PEEP in the low tidal volume group. In addition, to maintain similar targets for $PaCO_2$, the low tidal volume group had much higher respiratory frequencies, resulting in the de-

velopment of intrinsic PEEP. Therefore, the role of elevated levels of end expiratory pressure (PEEP) on survival of the low tidal volume group may have been obscured. Despite improved survival with the low tidal volumes group, survival was less than that of Amato's[16] combined approach (tidal volume reduction and PEEP elevation). As a result, the planned ARDSNet Assessment of Low tidal Volume and Elevated end-expiratory volume to Obviate Lung Injury (ALVEOLI) study will evaluate the role of higher levels of PEEP on survival. ARDSNet ALVEOLI will use data from the pressure-volume curve to develop the PEEP scale (PEEP scale = fraction of inspired oxygen:PEEP).

However, recent data suggest that determining optimal PEEP from the pressure-volume curve may be inaccurate.[17] In addition, recruitment to prevent cyclic airway closure (low volume lung injury) requires pressure in excess of 30 cm of water pressure. Complete recruitment exceeds the lower inflection point used by Amato et al.[16] to determine optimal PEEP levels. Recruitment begins at the lower inflection point and continues to the upper inflection point.[18–20] Therefore, elevated baseline airway pressure during APRV may produce near complete recruitment, thus minimizing low volume lung injury from cyclic recruitment. Additionally, APRV is less likely to produce over-inflation or high-volume lung injury, as airway pressures are lowered (released) to accomplish ventilation.

Other lung protective strategies include optimization of current modes of ventilation and alteration of ventilator strategies to prevent or reduce ventilator-associated lung injury. Current goals of ventilation include the following:

- avoiding extension of lung injury,
- minimizing oxygen toxicity by using mean airway pressure (P_{aw}),
- recruiting alveoli by raising mean P_{aw} by increasing PEEP and/or prolonging inspiration,
- minimizing peak P_{aw},
- preventing atelectasis, and
- using sedation and paralysis judiciously.[21]

Although first described 11 years earlier,[1,2] APRV may have benefits for preventing or limiting ventilator-associated lung injury.

☐Terminology

Unfortunately, a consistent vocabulary for APRV has failed to mature. Four commonly used terms include: pressure high (P High), pressure low (P Low), time high (T High), and time low (T Low).[7] P High is the baseline airway pressure level and is the higher of the two airway pressure levels. Other authors have described P High as the CPAP level,[22] the inflating pressure,[23] or the P1 pressure (P1). P Low is the airway pressure level resulting from the pressure release. Other authors may refer to P Low as the PEEP level,[22] the release pressure,[23] or the P2 pressure (P2). T High corresponds with the length of time for which P High is maintained; T Low is the length of time for which the P Low is held (i.e. for which the airway pressure is released).

The mean airway pressure can be calculated as follows:[7]

$$\frac{(\text{P High} \times \text{T High}) + (\text{P Low} \times \text{T Low})}{\text{T High} + \text{T Low}}$$

Some ventilators may compute this automatically, making manual calculation redundant. Common terms associated with APRV are summarized in Table 2 and Figure 3.

Somewhat confusing to the understanding of APRV have been the subsequent descriptions of modes of ventilation that appear very similar to it. Biphasic positive airway pressure (BIPAP)[24,25] differs from APRV only in the timing of the upper and lower pressure levels. In BIPAP, T High usually is shorter than T Low. One description of BIPAP[25] subdivides it into four categories, one of which is APRV-BIPAP.

Intermittent mandatory pressure release ventilation (IMPRV),[26] another mode of ventilation similar to and sometimes confused with APRV, synchronizes the release event with the patient's spontaneous effort. The release occurs after the patient's second, third, fourth, fifth, or sixth spontaneous breath. Further, all spontaneous breaths are pressure supported to overcome the resistance associated with breathing through the endotracheal tube and ventilator tubing. Synchronization does not occur with the raising of airway pressure, only the release. Because the concept of dyssynchrony in APRV has not been demonstrated clearly—and has

TABLE 2 ■ Summary of Airway Pressure Release Ventilation Terminology

Term	*Definition*	*Alternative Names*	*Units of Measure*
Pressure High (P High)[7]	Baseline airway pressure level Higher of the two airway pressures	CPAP level,[22] Inflation pressure,[23] P1	Cm H_2O
Pressure Low (P Low)[7]	Airway pressure level resulting from pressure release. The lower of the two airway pressures	PEEP level,[22] Release pressure,[23] P2	Cm H_2O
Time High (T High)[7]	Length of time for which P High is maintained	T1	Seconds
Time Low (T Low)[7]	Length of time for which P Low is maintained	T2	Seconds
Mean P_{aw}	$\frac{(P\ High \times T\ High) + (P\ Low \times T\ Low)}{(T\ High + T\ Low)}$[7]	—	Cm H_2O

CPAP = continuous positive airway pressure; Cm H_2O = centimeters of water; PEEP = positive end expiratory pressure; P_{aw} = airway pressure.

been stated not to be an issue—the necessity of intermittent mandatory pressure release ventilation is questionable.[10]

Intermittent CPAP[27] is based on the principles of APRV but is intended for patients undergoing general anesthesia. Continuous positive airway pressure is applied at a level that will provide an adequate tidal volume, then removed for 1 second to produce tidal ventilation, then reapplied. Unlike APRV, intermittent CPAP is not intended to restore normal functional residual capacity or improve oxygenation, and it can be discontinued abruptly.

BiLevel ventilation[28] is defined as augmented pressure ventilation that allows for unrestricted, albeit pressure-supported, spontaneous breathing throughout the ventilatory cycle. Although similar to APRV, it incorporates the option of pressure support in the airway pressure waveform to augment spontaneous breathing.

☐Indications for APRV

Airway pressure release ventilation was designed to oxygenate and augment ventilation for patients with ALI or low-compliance

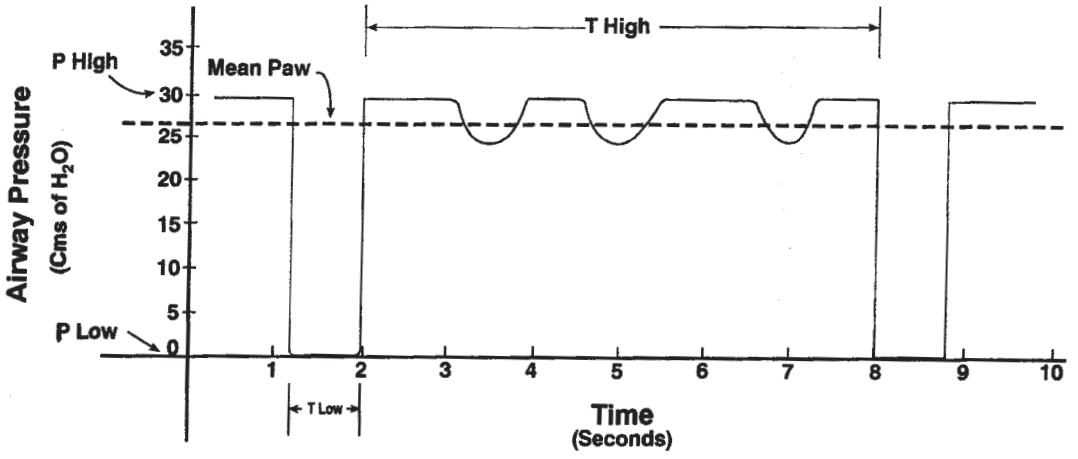

Figure 3. *Airway pressure release ventilation terminology. P_{aw} = airway pressure; P High = 30 centimeters of water (cms of H_2O); P Low = 0 cms of H_2O, T High = 6.0 seconds; T low = 0.8 seconds; calculated mean P_{aw} = 26.5 cms of H_2O.*

lung disease.[1,5,6] Airway pressure release ventilation also has been used successfully with patients with airway disease. Similar to CPAP, APRV can unload inspiratory muscles and decrease the work of breathing associated with chronic obstructive pulmonary disease.[29] Unlike PEEP (an expiratory flow resistor, which decreases expiratory flow), peak expiratory flow rates are increased during the release phase of APRV, improving expiratory flow limitation. Furthermore, during APRV, exhalation is not limited to the release phase, as it is permitted throughout the respiratory cycle.

The main causes of hypoxemia associated with ALI are shunting due to alveolar collapse and reduction in functional residual capacity.[1,7,30] Therefore, a primary goal of the treatment of ALI is recruitment of alveoli and prevention of derecruitment. Sustained plateau pressure is used to promote alveolar recruitment, while being maintained at an acceptable level. In addition, the number of respiratory cycles is minimized to prevent both the repetitive opening of alveoli and alveolar stretch, that may result in lung injury.

Patients in early-phase ALI often do not have impaired respiratory muscle strength or inadequate respiratory drive. Frequently, CPAP alone is sufficient to restore lung volume and increase lung compliance. However, when assistance with ventilation is required, APRV can be used. Intermittent airway pressure release allows alveolar gas to be expelled via natural lung recoil.[1]

□ Importance of Collateral Channels of Ventilation

Maintaining a constant airway pressure may be advantageous for several reasons. Constant airway pressure facilitates alveolar recruitment; enhances diffusion of gases; allows alveolar units with slow time constants to fill, preventing over-distension of alveoli; and augments collateral ventilation.[31]

Van Allen et al[32] noted that complete obstruction of an airway unit did not always result in collapse of the alveoli and, therefore, hypothesized that alternative pathways must exist. The pores of Kohn, located in the septa of the alveoli and open only during inspiration,[33] first were believed to be responsible. However, two additional pathways were later credited with playing a role: (1) Lambert's canals connect terminal and respiratory bronchioles with adjacent peribronchial alveoli, and (2) channels of Martin interconnect respiratory bronchioles and serve to bypass the main pathway (Figure 4).[34]

In normal, healthy lungs, collateral ventilation may barely occur at the functional residual capacity level, i.e. end exhalation. However, alternative pathways may be opened at a higher lung volume.[35] The role of alternative pathways in healthy lungs is very limited; but in disease states may be im-

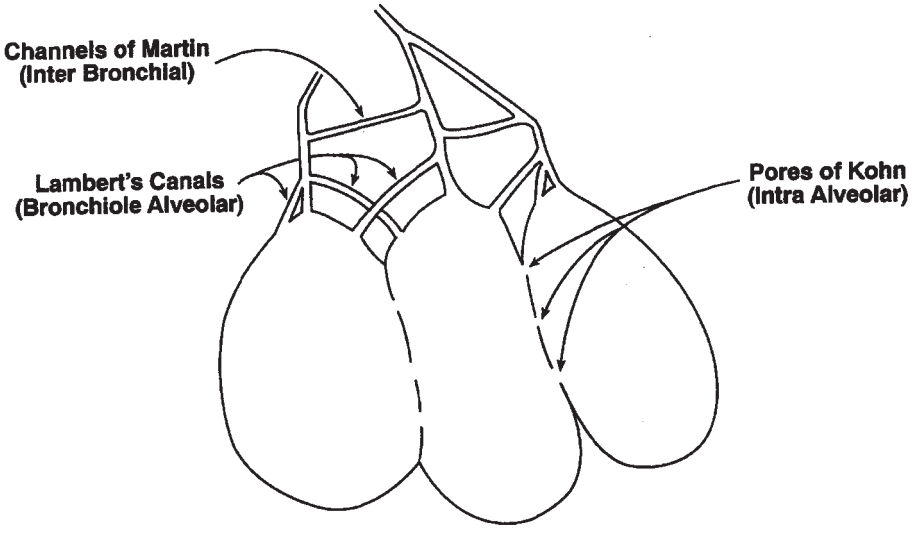

Figure 4. *Collateral channels of ventilation.*

portant.[36] Although collateral ventilation is typically lost in pulmonary edema, collateral pathways may be reopened and oxygenation improved by increasing functional residual capacity.[36] Sustained airway pressure, rather than intermittent periods of airway pressure, is more beneficial in the edematous, collapsed lung. Sustained breaths maintain a constant airway pressure and allow collateral channels to assist in producing ventilation. Collateral ventilation efficiency drops as respiratory frequency increases.[37] Airway pressure release ventilation uses these concepts, maintaining sufficient airway pressure for an adequate duration to open collapsed alveoli, thus improving recruitment of alveoli and increasing oxygenation.

□ Advantages

In patients with severe acute respiratory failure, the use of APRV results in significantly lower peak P_{aw}, when compared with continuous positive pressure ventilation (CPPV). Lower airway pressures are thought to be associated with a reduced risk of ventilator-associated lung injury.[14] Further, APRV requires lower minute ventilation than CPPV, suggesting less dead-space ventilation.[23] Studies of patients with ALI have shown that APRV supports oxygenation and ventilation, while producing lower peak P_{aw} than volume assist-control ventilation[5] and intermittent mandatory ventilation.[8,11] Similarly, animal studies of injured lungs suggest lower airway pressure, reduced dead space ventilation, and improved oxygenation and ventilation, when compared with intermittent positive pressure ventilation.[2]

Airway pressure release ventilation recruits lung units by optimizing end-inspiratory lung volume. Ideally, the end-inspiratory pressure, which equates to P High or plateau pressure, should be kept beneath 35 cm of water pressure.[9] This protective lung strategy has several positive effects. First, the preset pressure limit prevents, or limits, over-distension of alveoli and high-volume lung injury. Second, APRV affects tidal ventilation by decreasing rather than increasing airway pressure. Decreasing lung volume for ventilation further limits air space over-distension and the potential for high-volume lung injury. Third, maintaining airway pressure optimizes recruitment and prevents or limits low-volume lung injury by avoiding the repetitious opening of alveoli.[14]

High-volume lung injury occurs as a result of tidal ventilation above the upper inflection point of the pressure-volume curve. Low-volume lung injury results from ventilation beginning beneath the lower inflection point.[17] Airway pressure release ventilation begins on the pressure-volume curve between these two points and uses a release, not an increase, of pressure from its baseline. Therefore, oxygenation and ventilation occur predominantly within the upper and lower inflection points (Figure 5).

Calzia and Radermacher,[38] in their 10-year literature review of APRV, were unable to document any severe adverse effects of APRV and BIPAP on cardio-circulatory function. One case report[39] demonstrated an increase in cardiac output and blood pressure when APRV was used. Further, the authors suggested that it should be considered as an alternative therapy to pharmacologic or fluid therapy in the hemodynamically compromised, mechanically ventilated patient.

Animal studies indicate that APRV does not compromise circulatory function and tissue oxygenation, whereas CPPV can impair cardiovascular function significantly.[40] Spontaneous ventilation has a positive effect on the venous thoracic pump mechanism. Suppressing spontaneous breathing during CPPV can compromise cardiac function by decreasing venous return, thus cardiac output.[4]

The main advantage of APRV is that it allows for spontaneous breathing to occur at any point in the respiratory cycle. Depending on the patient's need, spontaneous breathing may involve only exhalation, only inspiration, or both.

The distribution of ventilation is significantly different when a spontaneous breath is compared with a mechanically controlled or assisted breath. Spontaneous breaths tend to improve ventilation-perfusion matching by preferentially aerating well-perfused, dependent lung regions. Mechanically delivered breaths primarily ventilate areas away from those receiving maximal blood flow. This phenomenon is consistent with earlier research, which demonstrated that spontaneous ventilation opens more alveoli, im-

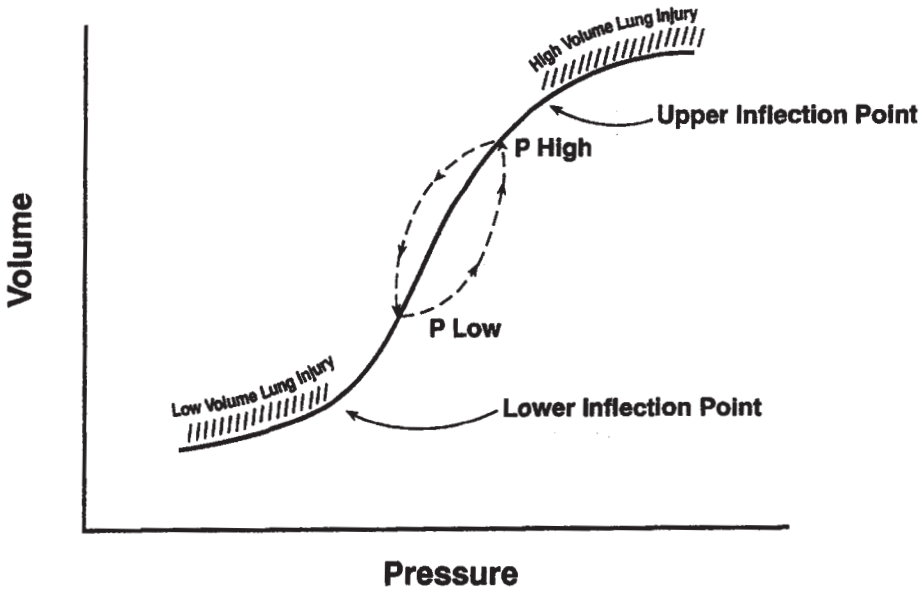

Figure 5. *Pressure-volume curve. Conceptual drawing of airway pressure release ventilation occurring below the upper inflection point and above the lower inflection point, achieving goals of lung protective strategies.*

proves regional gas exchange, and reduces atelectasis.[41]

Putensen et al.[42,43] found that by allowing unsupported, spontaneous breathing (using BIPAP or APRV) in both dogs[42] and humans[43] with ALI, ventilation-perfusion matching improved, as seen by a marked decrease in intrapulmonary shunt. In humans, however, pressure support ventilation preferentially ventilated poorly or nonperfused lung units that already were well ventilated. Furthermore, pressure support ventilation did not convert shunted areas to normal ventilation-perfusion units.[43]

Decreased need for sedation use or neuromuscular blockade use with APRV[7,8] and BIPAP[25] has been reported. Judicious use of sedation and paralysis in the mechanically ventilated patient was recommended at the American-European Consensus Conference on ARDS.[21] Unintentional, prolonged paralysis is now recognized as a complication of the long-term use of paralytics. In addition, a paralyzed diaphragm moves very differently with positive pressure ventilation compared with an active contraction. The paralyzed diaphragm is displaced preferentially along the path of least resistance, that is, into the abdomen of the non dependent region. This displacement leads to favored ventilation of the nondependent lung regions.[41] All of this contributes to both ventilation-perfusion mismatch and possible over-distension of healthy alveoli, leading to further hypoxemia (Table 3).

□ Disadvantages

Consistent with other pressure-targeted modes of ventilation, APRV is affected by changes in lung compliance and/or resistance. Clinicians need to identify the scenarios that affect lung volume and monitor patients for changes in their tidal volumes.

Because APRV is time-cycled, synchrony with the patient's spontaneous respiration does not occur. If a release phase is not synchronous with the patient's effort, discomfort may result. However, because APRV has a dynamic pneumatic system, inspiration and exhalation are facilitated at any time. Dyssynchrony with APRV has not been identified as a problem in the majority of the literature to date.[5,6,11]

As with any new technology, staff stress and subsequent increased risk to the patient may be noted with the implementation of APRV. Adequate and appropriate on-site training, coupled with off-site support services and backup, will help resolve some of the stress and decrease the risks associated with the in-

troduction of APRV. Transferring patients to subacute areas as their disease processes improve, may cause these issues to be revisited. Further, these areas may not have access to ventilators capable of delivering APRV, which will require switching the patient to a different mode of ventilation. Similarly, traveling to other departments (e.g., radiology, hyperbaric oxygenation chamber) may require temporary discontinuation of APRV, causing undue anxiety or discomfort in some patients.

Finally, only limited research exists regarding the clinical practice of APRV and its comparison with other modes. For example, APRV is suitable for ventilator weaning, though its superiority to mainstream modes, e.g. pressure support ventilation, has not been demonstrated. Weaning, in general, lacks a consensus, and this absence of absolutes exemplifies the great confusion within clinical practice and within the study of mechanical ventilation (Table 3).

TABLE 3 ■ Advantages and Potential Disadvantages of Airway Pressure Release Ventilation

Advantages
1. Lower P_{aw} for a given tidal volume compared with volume-targeted modes, e.g., AC, SIMV
2. Lower minute ventilation, i.e., less dead space ventilation
3. Limited adverse effects on cardio-circulatory function
4. Spontaneous breathing possible throughout entire ventilatory cycle
5. Decreased sedation use
6. Near elimination of neuromuscular blockade use
Potential Disadvantages
1. Volumes change with alteration in lung compliance and resistance
2. Process of integrating new technology
3. Limited access to technology capable of delivering APRV
4. Limited research and clinical experience

P_{aw} = airway pressure; A-C = assist control; SIMV = synchronized intermittent mandatory ventilation; APRV = airway pressure release ventilation.

□ Application of APRV

Little direction on the application of APRV can be found in the literature, other than as suggested by vendors and limited study protocols. However, based on an understanding of pulmonary physiology and pathophysiology, coupled with the theoretical understanding of mechanical ventilation[44] and current recommendations from consensus conferences,[9,21] the following technique has evolved.

When changing a patient's mode of ventilation to APRV, the initial settings are partly deduced from values of conventional ventilation. The clinician converts the plateau pressure of the conventional mode to P High and seeks an expired minute ventilation of 2 to 3 L/minute, less than when on conventional ventilation. This is accomplished by setting P High at the plateau pressure, with a ceiling level for the P High normally at 35 cm of water pressure. P Low is set at 0 cm of water pressure. A P Low of zero produces minimal expiratory resistance, thus accelerating expiratory flow rates, facilitating rapid pressure drops. T High is set at a minimum of 4.0 seconds. A T High of less than 4.0 seconds begins to impact mean P_{aw} negatively. T Low is set between 0.5 and 1.0 seconds (often at 0.8 seconds). With these settings (P High = 35 cm of water pressure, P Low = 0 cm of water pressure, T High = 4.0 seconds, T Low = 0.8 seconds), the mean P_{aw} will equal 29.2 cm of water pressure. It is not possible for conventional volume targeted modes to maintain a mean P_{aw} of 29 cm of water pressure and limit the peak or plateau pressures to 35 cm of water pressure, and still produce sufficient tidal ventilation.[44]

Application of APRV to newly intubated patients usually involves using standard parameters and adjusting the settings accordingly. Commonly, in the patient with moderate to severe ALI we default to P High/P Low of 35/0 cm of water pressure and T High/T Low of 4.0/0.8 seconds and allow spontaneous breathing to take place.[44]

When attempting to avoid alveolar overdistension, the clinician must be cognizant of the plateau pressure, as this is the best clinically available estimate of average alveolar pressure.[9] Although based primarily on animal data, a plateau pressure (or P High) greater than 35 cm of water pressure is associated with lung injury and, therefore, should be kept beneath this level.

Rarely, an elevated P High (40–45 cm of water pressure) may be indicated, especially

for patients with low-compliance respiratory systems, (e.g., individuals with morbid obesity, abdominal distension, or chest wall edema), either for the purpose of oxygenation or ventilation.[44] Although not optimal, the increased P High would be less than the pressure generated by conventional modes to produce a similar response.[22]

The P Low of zero is selected because minimal resistance to exhalation is the goal. Higher pressures may impede expiratory gas flow during passive lung recoil. The valid concern of collapsing alveoli with a P Low of zero is negated with the use of a short T Low (0.5–0.8 seconds) to maintain end expiratory lung volume.

The minimum T High duration is 4.0 seconds. The goal is to create a nearly continuous airway pressure level, which serves to recruit collapsed alveoli and maintain recruitment, thus optimizing oxygenation and compliance. As a patient's lung mechanics improve, T High is progressively lengthened to 12 to 15 seconds, usually in 0.5 to 2.0 second increments.[44] A further advantage of the long T High is the reduction in the number of opening and closings of the small airways, one of the mechanisms implicated in the development of iatrogenic ALI.[14]

The T Low probably is the most closely studied of the 4 parameters. Early writings[5,22] suggested a T Low of 1.5 seconds as the norm, which allows for complete emptying of the lungs. A longer T Low (3.0–4.0 seconds) in animals with ALI was associated with a decrease in arterial oxygenation and the accumulation of hemorrhagic fluid in the endotracheal tube.[5] An excessively long T Low encourages alveolar derecruitment, atelectasis, and airway closure during the release phase. Alternatively, an insufficient T Low potentially may result in inadequate exhalation, leading to dead space ventilation, hypercapnia, and hemodynamic compromise.[45] Indeed, an appropriately timed T Low is vital.[44]

Optimal release time allows for adequate ventilation while minimizing lung volume loss. Essentially, release time should impede complete exhalation in the slower compartments of the lung (i.e., areas of high compliance or high resistance to exhalation) and generate regional intrinsic PEEP. Theoretically, this will enhance alveolar recruitment.[4,7]

Calculation of T Low depends on expiratory time constants (T), which are a product of the compliance of the respiratory system (CRS) and the resistance of the airways (R_{AW}); that is, $T = C_{RS} \times R_{AW}$.[4,45] Low-compli-

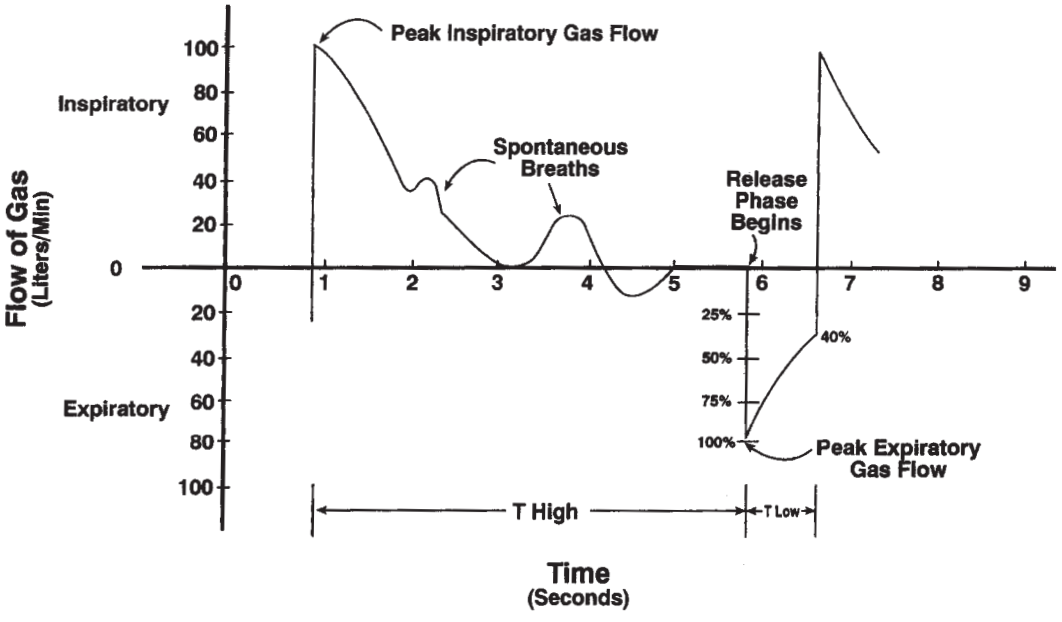

Figure 6. *Inspiratory and expiratory flow of gas in airway pressure release ventilation.[44] In this example, T Low terminates at 40% of the peak expiratory gas flow. Baseline airway pressure is then rapidly re-established. T High = 6.0 seconds; T Low = 0.8 seconds.*

TABLE 4 ■ Example of Airway Pressure Release Ventilation Settings in an Uncomplicated Case of Acute Lung Injury[43]*

P High (cm H_2O)	*T High (seconds)*	*P Low (cm H_2O)*	*T Low (seconds)*	*Calculated Mean Airway Pressure (cm H_2O)*
35	4.0	0	0.8	29.2
33	4.5	0	0.8	28.0
30	5.0	0	0.8	25.9
28	5.5	0	0.8	24.4
26	6.0	0	0.8	22.9
23	7.0	0	0.8	20.6
20	8.0	0	0.8	18.2
18	10.0	0	0.8	16.7
15	12.0	0	0.8	14.1

*Following the final settings, the patient was transitioned to CPAP of 12 cm of water pressure.

CPAP = continuous positive airway pressure; Cm H_2O = centimeters of water; P High = pressure high; T High = time high; P low = pressure low; T low = time low.

ance states, such as ARDS, will have lower (or shorter) expiratory time constants and therefore a lower (or shorter) T Low. High resistance diseases, such as asthma, will have longer time constants and require longer release times.[45] Determining the correct multiple of time constants to calculate T Low is a challenge of future research.

In practice, however, the clinician does not calculate the time constants for each patient, but rather relies on an approximation of the restriction of expiratory flow, as indicated by the expiratory flow of gas waveform (Figure 6). When expiratory flow falls to approximately 25% to 50% of peak expiratory flow, the clinician stops the release time and allows the airway pressure to return to P High.[44]

The transition to APRV may not result in instant improvement in oxygenation. Consistent with observations of inverse ratio ventilation,[4] the positive effects may take several hours to be realized. It appears that the recruitment of alveoli occurs "one by one." Sydow et al.[7] demonstrated that the maximal beneficial effect of APRV upon oxygenation occurred 8 hours after implementation, with no further improvement after 16 hours. In earlier studies, data were collected within the first 60 minutes after transition to APRV and thus the full effect of time on alveolar recruitment was not appreciated.

☐ Weaning From APRV

The current technique of weaning from APRV is guided by general principles of weaning used in clinical practice today. Knowledge of the signs of respiratory failure, as well as exclusion or correction of contributing factors preventing successful weaning, such as excessive secretions, bronchospasm, sepsis, anxiety, and diameter of endotracheal tubes and other dead space devices, are paramount. The approach in APRV is to maintain lung volume, improving both oxygenation and ventilation. As such, rarely does a specific point in time occur when weaning is "officially" commenced.

Primarily, the method to reduce support is through manipulation of P High and T High. P High will be lowered 2 to 3 cm of water pressure at a time, and T High will be lengthened in 0.5- to 2.0-second increments, depending on patient tolerance. The goal is to arrive at straight CPAP—usually at 12 cm of water pressure—and then the clinician either weans CPAP or simply extubates the patient at 6 to 12 cm of water pressure. Before switching to CPAP, P High often is approximately 14 to 16 cm of water pressure and T High is at 12 to 15 seconds (Table 4).[44] Patients with more severe forms of ALI or ARDS are weaned on a slower basis. Changes in mean P_{aw} are monitored closely for their effect on oxygenation. Simi-

larly, exhaled minute ventilation is tracked in conjunction with CO_2 removal.

□Conclusion

Airway pressure release ventilation can maintain oxygenation and ventilation at a level comparable to CPPV. Airway pressure release ventilation is associated with significantly lower peak airway pressures and dead space ventilation. Airway pressure release ventilation uses almost constant airway pressure that not only facilitates alveolar recruitment but also sustains that recruitment once it has occurred. Spontaneous, unsupported breathing during APRV may occur at any point in the ventilatory cycle. Spontaneous breathing is advantageous because it decreases intrapulmonary shunting and improves venous return. The ability to avoid neuromuscular blockade and decreased use of sedation have resulted in fewer complications and decreased drug costs. Finally, ventilator-associated lung injury, which can result from both high- and low-volume lung ventilation, may be balanced and averted.

Few clinicians believe that any single, isolated treatment can be responsible for a major improvement in the outcome for patients with ARDS. Combination therapy is expected to be the standard, including such concepts as prone positioning and permissive hypercapnia. Part of that therapy may include the ventilator strategy of APRV, which incorporates the advantages listed above. The authors believe that future research will support the use of APRV as the mode of choice for patients with ALI and ARDS.

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