Airway Pressure Release Ventilation

A Field Guide for the Emergency Physician

Rory Spiegel, MD[a,b,*], Max Hockstein, MD[a,b,1]

KEYWORDS

• APRV • ARDS • Hypoxemia

KEY POINTS

- Airway pressure release ventilation (APRV) uses high airway pressures to recruit and maintain lung volumes.
- The goals of APRV are to return patients lung volumes to functional residual capacity and to promote safe spontaneous breathing.
- APRV should be viewed as continuous positive airway pressure with release volumes intended to augment ventilation.

INTRODUCTION

The goal of mechanical ventilation (MV) is to provide a critically ill patient with physiologic gas exchange, whereas minimizing iatrogenesis and facilitating ventilator liberation.[1] Substantial effort has been placed into defining safe MV parameters for patients with and without acute respiratory distress syndrome (ARDS).[2,3] Owing, in part, to dedicated study, the mortality of ARDS has improved during the past several decades.[4] Salient discoveries in the management of ARDS include the institution of lung protective ventilation and prone positioning.[2,5] Benefits common to both of these interventions include the avoidance of overdistension of healthy alveoli and the maintenance of lung volumes.

CASE PRESENTATION

A 49 year-old woman is admitted to the ED for pneumonia. During her ED course, she becomes more hypoxic requiring endotracheal intubation. Unfortunately, there are no ICU beds available. A chest X-ray demonstrates bilateral, patchy infiltrates with

[a] Department of Emergency Medicine, MedStar Washington Hospital Center; [b] Department of Critical Care, MedStar Washington Hospital Center
[1] Present address: 524 8th Street Northwest, Washington, DC 20002.
* Corresponding author. 1423 33rd Street Northwest, Washington, DC 20007.
E-mail address: rspiegs@gmail.com

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adequate positioning of the endotracheal tube. The patient was placed on volume control ventilation with a rate of 24, a tidal volume of 500 mL, an Fio_2 of 0.8, and a PEEP of +10 cm H_2O. A plateau pressure was measured at 28 cm H_2O. An ABG reveals a pH of 7.34, a Pco_2 of 57 mm Hg, and a Po_2 of 50 mm Hg.

ACUTE RESPIRATORY DISTRESS SYNDROME PHYSIOLOGY

ARDS is a process, which reduces the size of the functional lung resulting in a "baby lung."[6] The "baby lung" is the island(s) of healthy lung parenchyma adjacent to the parenchyma affected by ARDS. MV exposes both the affected and unaffected lung to the set ventilation parameters; however, the 2 parenchymal areas accommodate gas flow differently. Delivered gas is directed preferentially to the alveoli with the lowest alveolar pressures, the unaffected regions of the lung are subjected to higher delivered volumes leading to overdistension and potential worsening of lung injury.[7–9]

Although ARDS has expansive causes and complex physiologic implications, in the ED, it is easiest to think of it as a portion of the lung as derecruited and, therefore, not participating in ventilation. Although ventilation is preferentially distributed to the nonaffected areas of the lung, perfusion is more homogenously distributed throughout the lung. Perfusion of nonaerated lung leads to blood passing through the lung without the opportunity to participate in gas exchange, a phenomenon known as shunt. Shunt is reflected by hypoexmia that seems to be refractory to the application of supplemental oxygen.

The more severe the ARDS, the larger the portion of affected lung. Consequently, the larger the portion of lung affected, the worse the hypoxemia. As the definition of functional residual capacity (FRC) is defined as "the volume of gas in the lung after a normal expiration," in patients with ARDS, the patient's FRC is significantly reduced.[10]

Alveolar recruitment does not follow a commonly conceived "inflated or deflated" paradigm. Instead, the lung exhibits viscoelastic behavior. Alveoli both dynamically resist volume changes over time (a property of viscosity) and quickly assume their derecruited form when the stressor is removed (a property of elasticity). Therefore, the lung does not instantaneously change its volume in response to the application of pressure; rather, it will do so over time. Given these physiologic properties, it stands that one must apply pressure-over-time in order to rerecruit lung and restore FRC. This is why tidal pressures do nothing to recruit lung and the application of PEEP is necessary to optimize alveolar recruitment and prevent derecruitment by raising mean airway pressures.

The goals of MV in patients with ARDS are 2-fold. First, to ensure the volumes of gas delivered is appropriate for the size of the lung participating in ventilation. Second, to restore patients' FRC, whereby increasing the portion of the lung, which is participating in ventilation.

AIRWAY PRESSURE RELEASE VENTILATION

Airway pressure release ventilation (APRV) is a mode of ventilation that uses high airway pressures to recruit alveoli and maintain patients' physiologic lung volumes. The goal of this mode of ventilation is 2-fold: first, to maintain patients as close to their FRC as possible and second, to promote safe spontaneous breathing. Although there are several unique names for the mode by different manufacturers (**Table 1**), all function similarly.

APRV should essentially be viewed as continuous positive airway pressure (CPAP), with intermittent releases of that pressure to metabolically support patients who are

Table 1
Airway pressure release ventilation mode names by different manufacturers

Manufacturer	Mode Name
Drager	PC-APRV
CareScape	APRV
Hamilton Galileo	APRV
Puritan Benett	840: BILEVEL 980: APRV
Servo	Bi-Vent
Vela	APRV/BiPhasic

Abbreviations: APRV, airway pressure release ventilation.

incapable of managing their ventilatory load. Imagine for a moment, a patient presents to you in the emergency department in respiratory distress. This patient was breathing comfortably with normal respiratory mechanics just a few days prior. Whatever respiratory insult that caused this presentation is simply due to worsening compliance of the lung parenchyma resulting in reduced resting lung volumes less than FRC. The patient's respiratory muscles are working just as well as they did previously, only now they have to work harder to manage the same volume of CO_2 production.

If one could restore the patient back to their previous lung volumes with the use of PEEP, then potentially the patient could more comfortably breathe on their own. The classic example of this is a patient presenting to the ED with pulmonary edema due to congestive heart failure. These patients typically have relatively normal respiratory muscles and have respiratory distress due to the increased extravascular lung water leading to a heavy lung resulting in reduced lung volumes. The use of noninvasive positive pressure ventilation is so effective in this patient population because it allows for the clinician to reestablish normal lung volumes using a noninvasive form of PEEP (expiratory positive airway pressure). Rapid restoration of normal lung volumes is readily obtainable due to the relative swift recruitability of patients with cardiogenic pulmonary edema. This is why most of these patients are managed noninvasively, not requiring invasive MV.

Unfortunately, most forms of respiratory distress are due to underlying disease processes that are not as easily recruitable as cardiogenic pulmonary edema. Moreover, although the application of PEEP will often restore normal lung volumes, it does so in a slow fashion. Given the reluctant nature of lung recruitment of an injured lung, it is not feasible or safe to expect patients to manage their own ventilation. During this period of recruitment, until the patient can safely manage their own ventilatory needs with spontaneous breathing, APRV uses what is known as a release volume to achieve its ventilatory ends.

A release volume consists of a momentary release of CPAP causing a rapid decrease in airway pressure and the subsequent exhalation of CO_2 (**Fig. 1**). What makes APRV different from other forms of inverse ratio ventilation (IRV) (where inspiratory-to-expiratory ratio exceeds conventional targets) is that this release volume is precisely controlled to encourage offloading of CO_2 while limiting the actual change in lung volumes. In fact, we would argue that it is the control of the release volumes, which endow APRV with its unique properties. Although there are many other aspects of APRV that will be discussed in this review, unless clinicians are mindful of preventing derecruitment during the release phases, then the benefits associated with this mode of ventilation are likely to be lost and additional injury may occur.

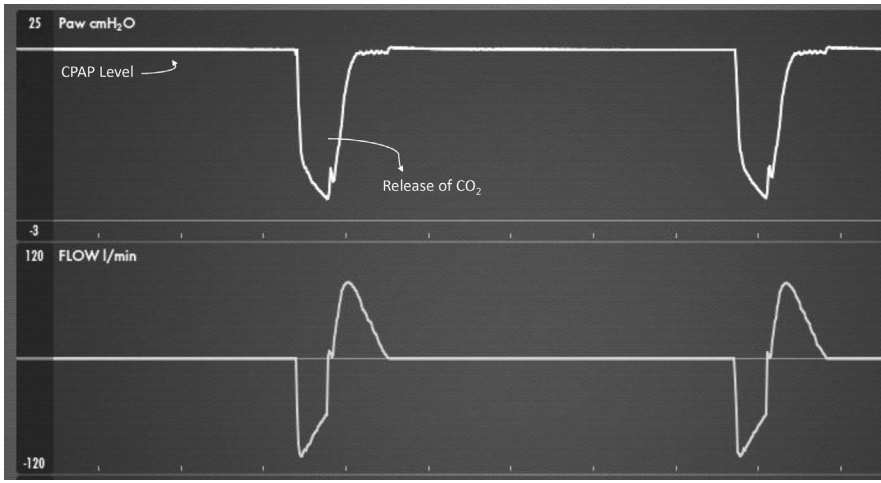

Fig. 1. APRV pressure and flow scalars demonstrating 2 phases: CPAP phase and release phase. During CPAP level, alveolar recruitment is accomplished and during the release phase, CO_2 is released.

As patients recruit and lungs approach the patient's natural lung volumes, their ability to breathe spontaneously and manage their own ventilatory needs improves. As such, the need to augment their ventilatory efforts decreases, and release breaths are required less frequently. Eventually, patients are able to fully support their ventilatory needs and no longer require any release breaths to maintain normal CO_2 levels. Now, the patients can be "stretched" to CPAP (**Fig. 2**).

When discussing the literature examining the efficacy of APRV, it is important to differentiate IRV from APRV. IRV is a pressure-controlled mode of ventilation that spends most of the respiratory cycle in the inspiratory phase and, as a consequence,

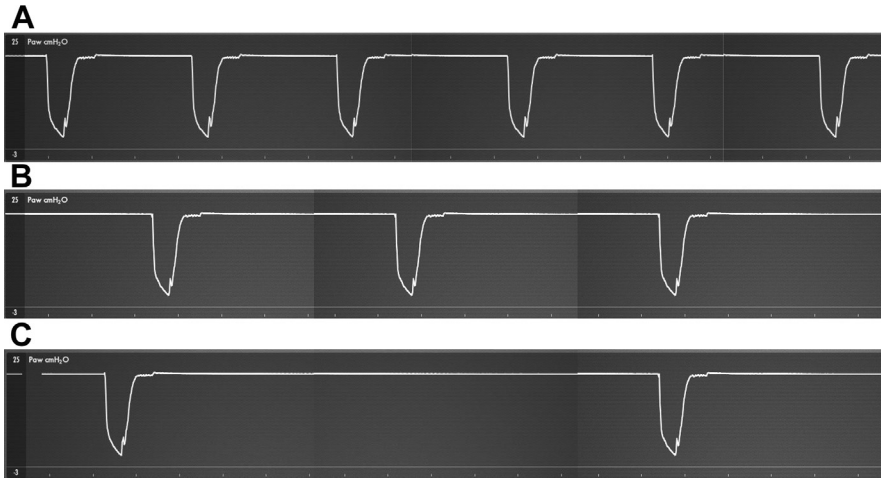

Fig. 2. APRV liberation. Note progressive elongation (stretching) of T-high from panels A through C. (*A*) initial APRV setup. (*B*) APRV maintenance. (*C*) Approaching evolution to CPAP.

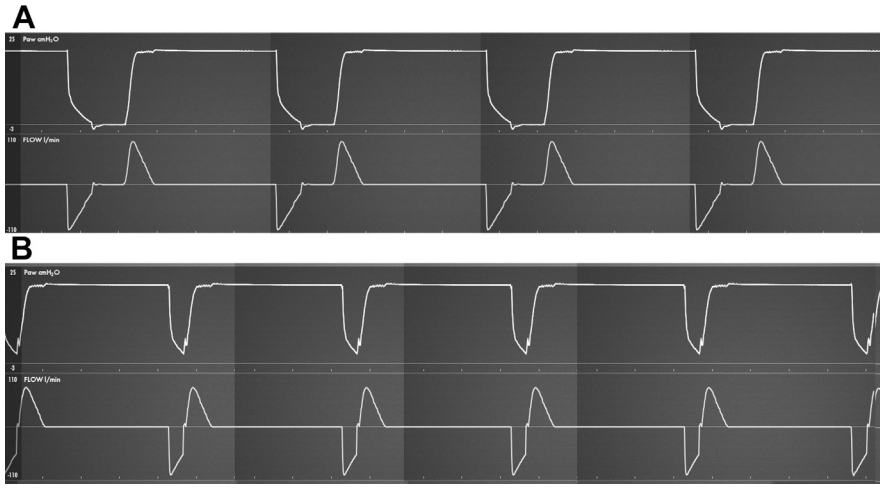

Fig. 3. (*A*) Note excessive timing of T-low resulting in full exhalation manifested by flow scalar returning to baseline between CPAP phases. (*B*) Appropriate timing of T-low resulting in trapping.

a far smaller portion in the expiratory phase. What differentiates APRV from the more general IRV, is the expiratory phase is titrated to the patient's individual lung compliance, ensuring that the expiratory time is limited to 75% of patient's peak expiratory flow rate (PEFR; **Fig. 3**). In other words, when compared with breaths delivered in traditional modes of MV, APRV does not allow for a "full" exhalation (ie, 100% of PEFR) during the timed release.

Jain and colleagues noted that there is an inconsistency among the studies claiming to study APRV because many of them use a fixed expiratory time (fixed APRV), not titrating based on the patient's individual lung mechanics (personalized APRV), thus allowing many of the patients to fully exhale.[11] When all studies claiming to examine APRV are examined in totality, there does not seem to be a benefit in its use but when the studies that do not mandate a titration of the time low (T-low) in concordance with the patient's lung mechanics are excluded, APRV was found to be superior to more traditional modes of ventilation, leading to a decrease in the time spent on MV as well as mortality.

Probably, the most well-done RCT comparing APRV to LTV strategies using more traditional modes of ventilation is a trial published by Zhou and colleagues.[12] Published in Intensive Care Medicine in November of 2017, the authors enrolled patients presenting to a single center with hypoxic respiratory failure, requiring mechanical ventilatory support, who fulfilled the diagnostic criteria of ARDS according to the Berlin definition, a Pa_{O_2}/Fi_{O_2} of less than 250, and were mechanically ventilated for less than 48 hours. Patients were randomized to receive either a traditional ARDSNet lung protective strategy (targeted tidal volume of 6 mL/kg predicted body weight using a volume assist mode of MV, PEEP levels determined by a PEEP-FiO_2 table and respiratory rates [RRs] titrated to limit hypercapnia and respiratory acidosis), or to APRV. Patients randomized to the APRV arm were placed on a pressure high (P-high) according to the plateau pressure that was obtained on their previous traditional volume control setting, a pressure low (P-low) of 0 to 5 cm H_2O, and a T-low titrated to the patients' intrinsic compliance.

Overall, the patients randomized to receive an APRV strategy did remarkably better than their low-tidal volume counterparts, boasting significantly fewer ventilator days (8 [5–14] vs 15 [7–22] $P = .001$), higher rate of successful extubation (66.2% vs 38.8% $P = .001$), and fewer tracheostomies (12.7% vs 29.9% $P = .013$). They also required less additional supportive measures including paralysis, recruitment maneuvers, and prone positioning. Moreover, although not statistically significant, the point estimate of both ICU mortality and hospital mortality trended strongly in favor of the APRV group (ICU mortality 19.7% vs 34.3% $P = .053$, hospital mortality 23.9% vs 37.3% $P = .088$).

Following the publication of the Zhou and colleagues trial, a meta-analysis by Lim and colleagues was published in Critical Care Medicine in 2019.[13] The authors identified 7 RCTs with a total of 412 patients admitted to the ICU for ARDS on MV and randomized to either APRV or a traditional mode of ventilation. The authors found an improvement in mortality in patients who received APRV compared with traditional modes of ventilation.

WHEN TO CONSIDER AIRWAY PRESSURE RELEASE VENTILATION?

Although APRV was once considered a "rescue" mode of MV, its early application in a patient's course has tangible benefits regarding lung mechanics and mortality. APRV has been examined across several clinical domains including ARDS from pneumonia, pulmonary contusions in the setting of trauma, and postcardiac surgery.[12,14,15] In the emergency department, APRV's primary role should be in patients with hypoxemia refractory to traditional modes of ventilation. For the sake of this document, we will consider refractory hypoxemia as an oxygen saturation less than 92% despite an Fio_2 of 1. Typically, cases of refractory hypoxemia in the ED are due to intrapulmonary shunting.[16]

Before using APRV in a patient with refractory hypoxemia, the clinician should endeavor to identify the type of shunt causing the hypoxemia. Clinicians will encounter 2 distinct types of intrapulmonary shunt leading to refractory hypoxemia, single or double lung shunt physiology.

Single lung shunt physiology occurs when the pathologic process is occurring in only one of the patient's lungs and leaves the contralateral side untouched. This can often be caused by lobar pneumonia, mucous plugging, atelectasis, and right mainstem intubation (**Fig. 4**A). Double lung shunt physiology occurs when the cause of the hypoxemia is more diffuse and affects bilateral lungs (**Fig. 4**B). This is often seen in multifocal pneumonia, pulmonary edema, ARDS, and so forth. Single versus double lung physiology can often be distinguished using a portable chest X-ray or point-of-care lung ultrasonography.

The importance of determining the source of a patient's shunt before intervening is that these distinct phenotypes respond differently to increases in mean airway pressure and thus tolerance to APRV. Most notably, in patients with single lung physiology, the application of a high pressure/PEEP ventilatory strategy will often lead to a paradoxic worsening of the patient's hypoxemia. This is due to a disproportionate application of pressure to the noninvolved lung leading to overdistention and worsening of the patient's shunt physiology.[17] Once the presence of single lung shunt physiology has been excluded as the source of the patient's refractory hypoxemia, APRV can be considered as an appropriate modality.

INITIAL SETTINGS AND TITRATION

APRV has 5 settings: P-high, P-low, time high (T-high), T-low, and fraction of inspired oxygen (Fio_2) (**Fig. 5**). Appropriate manipulation of these settings will optimize

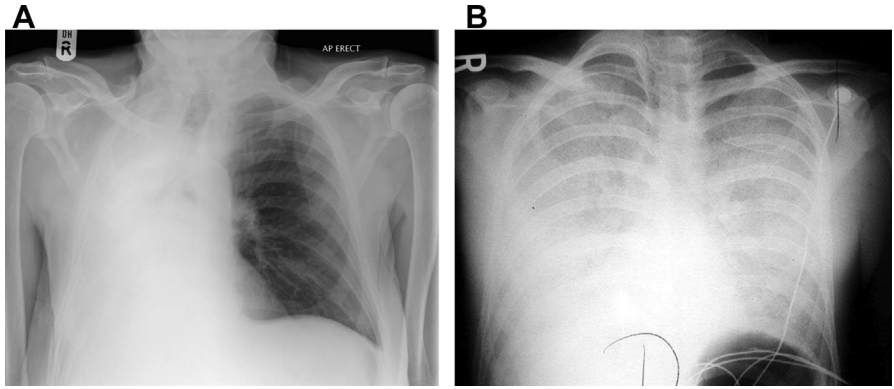

Fig. 4. (*A*) Single lung shunt physiology from complete atelectasis of the right lung causing "whiteout." (*B*) Double lung shunt physiology as manifested by opacification of both lung fields.

oxygenation, ventilation, and pulmonary mechanics for patients on APRV. It is important to keep in mind that the goal with APRV is to return patients to FRC and encourage safe spontaneous ventilation.

P-high: Pressure-high is analogous to CPAP. P-high is the maximum and sustained pressure that the ventilator will deliver during which the patient should spontaneously breathe. An appropriately set P-high will be at a pressure, which restores the patient's FRC, enabling safe, spontaneous ventilation in addition to optimal ventilation-perfusion matching. Notably, spontaneous ventilation that occurs at a higher PEEP results in more homogeneous parenchymal distension.[18]

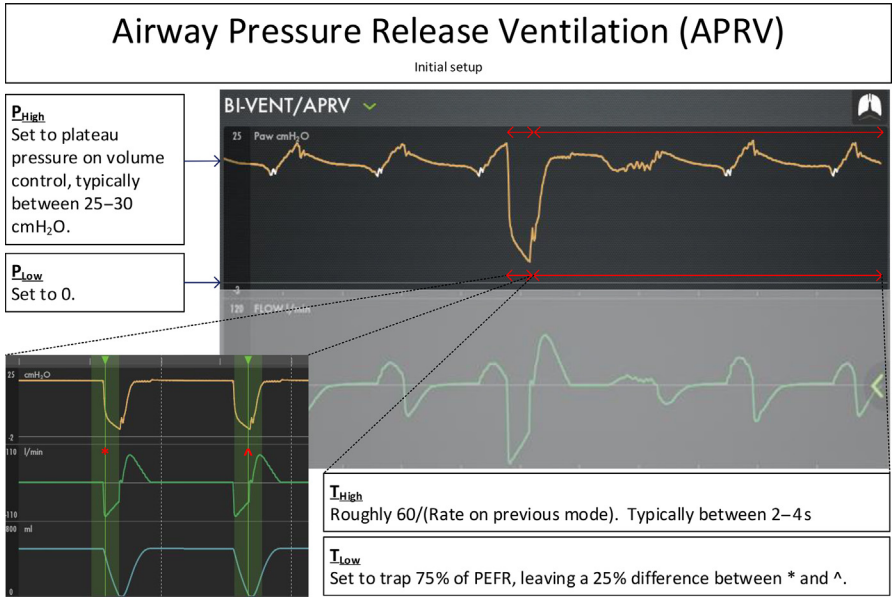

Fig. 5. APRV pressure and flow scalars with recommended settings.

P-high should be set at approximately the patient's plateau pressure, typically starting within 2 cmH_2O of the plateau pressure. If the patient's hypoxia worsens when initially placed on APRV, it is possible that the alveoli have distended to the point of collapsing adjacent blood vessels, preventing oxygenated blood from returning to the left heart. In this scenario, lowering P-high by 2 cmH_2O is reasonable. Increasing the value of P-high should be undertaken if clinicians suspect underdistension as the cause of continued hypoxemia.

T-high: The time the patient spends at the P-high between release volumes is the T-high. Typically initial values for P-high range from 25 to 35 cmH_2O. T-high typically ranges from 3 to 5 seconds but in cases of severe ARDS, it can be as low as 1.5 seconds. The initial goal should be to approximate the patient's minute ventilation on traditional modes of ventilation. The total time of a single respiratory cycle in APRV is the sum of T-high and T-low. Therefore, 60/(T-high + T-low) = RR. Because T-low is commonly fairly low, this formula can be simplified to 60/T-high = RR. Typically, in the ED, it is recommended to set the T-high to 2 seconds and titrate to match the patients MV requirements.

P-low: Pressure-low is the minimum pressure achieved in the respiratory cycle, typically set to 0 cmH_2O. It is important to note that the P-low is set to 0 cmH_2O to achieve the greatest pressure differential between the P-high and P-low to encourage both high peak expiratory flow rates and bulk ventilation. Although the ventilator decrements to a pressure of 0 cmH_2O, the patient's alveolar pressure does not. It is not the P-low that prevents derecruitment in APRV but rather the time during which the ventilator is set to stay at P-low before reengagement of the P-high. Remember, the P-low is used to achieve maximal expiratory flow rates. The T-low is the setting, which prevents derecruitment.[19] As such, even if the P-low on the ventilator is set to zero, as long as the T-low is set properly, the alveolar pressure is unlikely to decrease to zero.

T-low: T-low is arguably one of the most important settings of APRV because it controls lung volumes during bulk ventilation and prevents derecruitment and limits atelectrauma. Patients with ARDS have alveoli that collapse faster than uninjured (or less injured alveoli). A T-low that is set to allow for full exhalation would allow for alveolar collapse and, therefore, derecruitment leading to atelectrauma. In other words, T-low, as opposed to P-low, is the variable that controls end-expiratory pressure. In a patient without spontaneous ventilatory efforts, during the ventilator transition (release) from P-high to P-low, the movement of gas (exhalation), known as release volumes, allows for ventilation. There is significant heterogeneity in how clinicians set T-low.[20] Commonly, T-low is set to trap 75% of a patient's expiratory flow, "creating" a pneumatic splint.

T-low should be individually titrated to the patient's expiratory flow curve with the goal of ending T-low at approximately 75% of the PEFR (**Fig. 6**). We recommend initially starting between 0.4 and 0.5 seconds and then making smaller titrations based on the patient's individual flow curves with the goal of trapping 75%.

Returning to the clinical vignette presented at the start of the article, despite attempts to recruit on traditional modes of ventilation, a follow-up blood gas revealed no improvement in hypoxemia. Given the refractory hypoxemia with double lung-shunt physiology, the decision was made to transition the patient to APRV with the following settings. The patient's plateau pressure was found to be 28 and so a P-high of 28 was selected. P-low was set as 0. Given the patients hypercarbia despite a RR of 24, a T-high 2.0 was selected. The T-low was set to 0.4 and was measured to appropriately trap 75% of PEFR. The Fio_2 was maintained at 1.0 with the intent to titrate down as the lung recruited during the next few hours.

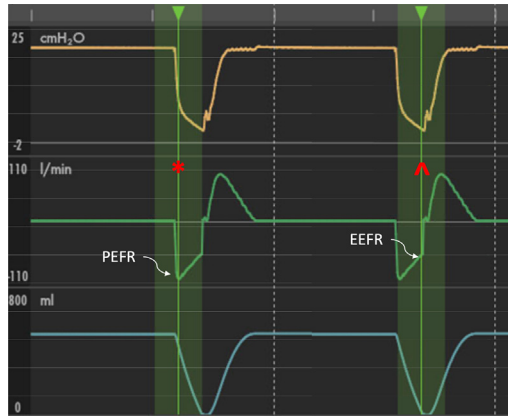

Fig. 6. APRV flow scalars demonstrating how to measure PEFR and EEFR in order to appropriately set T-low.

Once the precipitant of the hypoxic respiratory failure resolves and appropriate physiology has been restored, patients can be liberated from the ventilator. The classic strategy for weaning from APRV is known as "drop-and-stretch." "Dropping" refers to sequential decrementing of P-high and "stretching" refers to the incremental increase of T-high. Whether dropping and stretching are done in sequence or in tandem has been subject of debate. With the eventual elimination of T-low (by virtue of lengthening of T-high), the mode will no longer resemble APRV, rather, it will be CPAP.

HEMODYNAMIC CONSIDERATIONS

It is often stated that the high airway pressures used with APRV may lead to a greater degree of hemodynamic instability when compared with more traditional modes of ventilation. The observation that high airway pressures decrease preload and increase pulmonary vascular resistance gives hemodynamic consequences to APRV biological plausibility; observational data would seem to suggest it is untrue. There have been several observational studies examining patients with ARDS ventilated using APRV, in whom pulmonary artery catheters were placed. The patients were switched from traditional ventilation to APRV, and the authors observed that all hemodynamic indices improved following the initiation of APRV, including an improvement in lactate clearance and a decrease in pressor requirements.[21,22] Zhou and colleagues observed similar results in addition to a decrease in heart rate and an increase in mean arterial pressure, despite the APRV arm having significantly higher mean airway pressures.[12]

VENTILATORY CONSIDERATIONS

There is a common misconception that there is an increased rate of hypercapnia due to a decrease in the overall minute ventilation associated with the use of APRV. Never mind the more important question of whether hypercapnia is harmful in ARDS, there are numerous models demonstrating that the use of APRV promotes alveolar recruitment, improves ventilatory efficiency and increases CO_2 removal, despite a decrease in the total minute ventilation.[23,24] In a randomized control trial performed by Zhou and colleagues, the patients in the APRV group had an almost identical P_{CO_2} to the patients in the low-tidal volume group (40.8 $\pm$ 7.3 and 42.3 $\pm$ 8.6, respectively), despite using a significantly reduced minute ventilation (6.86 $\pm$ 2.06 vs 8.22 $\pm$ 2.30).

Although over time patients on APRV have lower minute ventilation requirements than patients on traditional modes of ventilation, this occurs gradually as patients improve their lung volumes. Initially, their minute ventilation requirements will be fairly close to what they were on traditional modes of ventilation. With that in mind, clinicians should endeavor to match the minute ventilation requirements previously required on standard modes of ventilation but should not be surprised if over time, these requirements decrease as patients' lung volumes begin to approximate FRC. For patients with continuous $EtCO_2$ monitoring, given the bulk of airflow occurs during T-low, the waveform will be the inverse of conventional waveforms. For patients on APRV, increasing $PETCO_2$ values may represent increasing P_{CO_2}, an increase in cardiac output, and an increasing number of alveoli now participating in ventilation.

MANAGING THE PATIENTS' RELEASE VOLUMES

Probably, the most frequent and strenuously used argument against the use of APRV is that it is often not in line with low-tidal volume ventilatory strategies and, thus, should be avoided in favor of a more traditional mode of ventilation where tidal volumes that are more precise can be obtained.

The ARMA trial published in the NEJM in 2001 examined the use of low-tidal volume ventilation in patients admitted to the ICU with ARDS. The authors found that patients randomized to a 6 cc/kg tidal volume strategy did significantly better than patients randomized to a 12 cc/kg strategy. Although this trial has a place in the tabernacle of critical care literature, the results are often misunderstood.

The ARMA trial found that patients admitted to the ICU with ARDS who were given an empiric 6 cc/kg tidal volume had a clinically significant reduction in mortality compared with patients who were given an empiric 12 cc/kg tidal volume. Or simply put, in patients with small absolute lung volumes (baby lung), a strategy that used empirically low tidal volumes was superior to a strategy that administered empirically high tidal volumes. The authors did not study whether an empiric 6 cc/kg tidal volume was optimal compared with the clinician at the bedside titrating the tidal volume to the patient's individual lung volume. Moreover, the ARMA trial was a trial examining 2 drastically different strategies of tidal volume. It did not in fact examine whether a specific mode of ventilation was optimal to any other mode of ventilation. Given the reality of ARMA, its methodology and its findings, it is nonsensical to extrapolate its findings as "proof" that a volume-cycled mode low-tidal volume strategy is superior to APRV.

A more genuine debate exists. APRV, at times, provides tidal volumes outside the safe volumes prescribed in the ARMA trial. Although release volumes may occasionally exceed 6 cc/kg, looking at tidal volumes in isolation lacks the nuanced understanding of APRV mechanics. The size of the release volume is determined by the P-high, T-low, and compliance of the patient's lung. In patients with poor compliance, due to severe ARDS, the size of the lung that is participating in ventilation is very low. In these patients, if the T-low is set to 75% of PEFR, then the release volume will also be low (often <6 cc/kg). As the patient's lung compliance improves, the release volumes will naturally increase without any change to either the P-high or the T-low. Essentially, the release volumes will be automatically tailored to the patient's individual, increasing functional lung size.

In patients with a severe reduction in lung volumes, who will truly benefit from LTV ventilation, APRV will deliver desired volumes. In patients with larger lung volumes, capable of accepting larger volumes, APRV will allow for these increased volumes. Thus, the debate should not be whether APRV is capable of delivering an LTV strategy in patients with ARDS but rather, in patients with improving compliance, is tolerating

the natural increases in release volumes safe? Although the data are not definitive, studies comparing APRV to traditional modes of ventilation have failed to identify any signals of harm due to the variable tidal volumes associated with the use of APRV. In fact, most of the preclinical data demonstrate decreased strain on the individual lung units due to APRV's superior recruitment capabilities.[25–27] At least, in its limited capacity, the Zhou and colleagues trial confirmed these findings. There was no statistical difference in the rate of pneumothoraces between the 2 groups, and the point estimate noticeably favored the APRV arm (4.2% vs 10.4% *P*-value of .199).

SUMMARY

Although the precipitant of respiratory failure is being managed, MV should optimize the patient's pulmonary and hemodynamic mechanics by way of restoration of the patient's FRC. By using higher than conventional mean airway pressures with APRV, FRC is restored, and physiologic breathing can resume.

CLINICS CARE POINTS

- Airway pressure release ventilation (APRV) has 5 settings: pressure high (P-high), pressure low (P-low), time high (T-high), time low (T-low), and fraction of inspired oxygen.
- P-high: Pressure-high is analogous to continuous positive airway pressure. P-high should be set at approximately within 2 cmH_2O of the plateau pressure.
- T-high: The time the patient spends at the P-high between release volumes. It is recommended to set the T-high to 2 seconds and titrate to match the patient's mechanical ventilation requirements.
- P-low: Pressure-low is the minimum pressure achieved in the respiratory cycle, typically set to 0 cmH_2O.
- T-low: T-low is arguably one of the most important settings of APRV because it controls lung volumes during bulk ventilation and prevents derecruitment and limits atelectrauma. We recommend initially starting between 0.4 and 0.5 seconds and then making smaller titrations based on the patient's individual flow curves with the goal of trapping 75%.

DISCLOSURE

The authors have nothing to disclose.

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