Airway Pressure Release Ventilation (APRV)

Practical Issues and Dräger Evita Series Set-Up

Definition:

- ❑ APRV is continuous positive airway pressure (CPAP) with regular, brief, releases in airway pressure
- ❑ As with CPAP, true APRV includes spontaneous breathing through an "open breathing system" or "floating exhalation valve system"
- ❑ APRV utilizes a release phase from an established CPAP level to supplement ventilation
- ❑ The CPAP level primarily facilitates oxygenation and the timed releases facilitate carbon dioxide clearance. Spontaneous breathing may occur at any time during the cycle enabling the patient to augment ventilation thought out the respiratory cycle.

Indications:

- ❑ APRV may be used for any patient requiring ventilatory support. APRV may be particularly beneficial in the treatment of patients with acute lung injury (ALI), acute respiratory disease syndrome (ARDS) and other forms of acute restrictive disease that involves recruitable lung elements
- ❑ Indicated as a single ventilatory mode capable of supporting severe respiratory failure and can be utilized continuously through to weaning

Contraindications:

- ❑ Patients with severe obstructive airway disease may be more complex to manage on APRV, but COPD is not an absolute contraindication
- ❑ Patients with significant bullous disease will require close observation for worsening of bullae

Benefits:

- ❑ The ability to breathe spontaneously throughout the entire ventilatory cycle
- ❑ Spontaneous breathing improves ventilation to poorly or non-ventilated lung regions reducing ventilation-perfusion mismatch
- ❑ During spontaneous, unassisted breathing, the posterior crura (posterior muscular) sections of the diaphragm will remain functional, minimizing posterior consolidation/atelectasis
- ❑ Patients in the supine position will have improved ventilation of dependent lung regions, reduced pulmonary shunt and improved oxygenation during spontaneous breathing
- ❑ However, if the diaphragm is paralyzed, the force of the abdominal contents will displace the diaphragm cephalad (towards the head) transferring intra abdominal pressure into the thorax enhancing dependent atelectasis. As a result, the machine delivered volume preferentially distributes to the more compliant, non-dependent lung (anterior) and less to the non-compliant, better perfused, dependent lung regions. Consequently, dead space ventilation and shunt are simultaneously worsened.
- ❑ Conversely, maintenance of diaphragmatic tone or inspiratory effort will minimize dependent atelectasis
- ❑ Lower peak airway pressures than conventional modes of ventilation
- ❑ More effective alveolar ventilation (superior recruitment) with resultant lower exhaled minute volume
- ❑ Enhanced effects on cardio-circulatory function
- ❑ Decreased need for patient sedation
- ❑ Significantly reduces or eliminates the need for neuromuscular blocking agents. Subsequently, immediate decrease in pharmacy expenses is noted. Eventually, other savings are incurred as the complications of prolonged neuromuscular blockade are avoided.

Potential Risks:

- ❑ Potential volume change with alteration in lung compliance and resistance.
- ❑ Patients with compliant lungs, e.g. COPD, may experience hemodynamic compromise and require a reduction in the mean airway pressure. Hemodynamic

instability, though not common, may occur in patients with intravascular volume depletion.

- ❑ In patients with compliant lungs (COPD), increase in mean airway pressure may cause increase in alveolar dead space, leading to elevated $PaCO_2$
- ❑ Learning curve associated with introduction of new technology

How Does It Work?

- ❑ APRV provides near continuous airway pressure to facilitate recruitment, lung volume maintenance, and stability, thereby enhancing oxygenation
- ❑ APRV produces tidal ventilation by briefly lowering airway pressure rather than elevating airway pressure (as with traditional ventilatory modes)
- ❑ Threshold (CPAP) valve facilitates spontaneous breathing and patient comfort throughout the entire respiratory cycle (patient controlled I:E ratio)
- ❑ Spontaneous breathing may occur at any period in the respiratory cycle

Initial Set-Up - General Guidelines for Adults:

<u>Transitioning a patient from conventional settings to APRV</u>:

1. Convert the plateau pressure of the conventional mode to P High. Normally, the maximum goal for P High is 35 cms H_2O. Limiting P High to 35 cms H_2O may minimize ventilator associated lung injury (VALI). However, a P High of >35 cms H_2O may be necessary in patients with decreased thoracic and abdominal compliance.
2. P Low is set at 0 cms H_2O, which produces minimal expiratory resistance and facilitates rapid pressure drops, thus accelerating expiratory flow rates.
3. T High is set at 4 to 6 seconds. T High of less than 4 seconds will begin to negatively impact mean airway pressure and recruitment.
4. T Low is typically set between 0.4 and 1.0 seconds (often 0.7 seconds) and is determined by analysis of the expiratory gas flow curve.

- ❑ Anticipate an expired minute ventilation of 2-3 L/minute less than when on conventional mode (less deadspace ventilation with APRV)
- ❑ Adjust settings based on patient's ABGs and clinical status

Application of APRV to Newly Intubated Patients:

- Common initial settings start at:
  1. P High 25-35 cms H_2O
  2. P Low 0 cms H_2O
  3. T High 4 to 6 seconds
  4. T Low 0.4-1.0 seconds

Reassessment / Subsequent Adjustments

- Adjust settings based on patient's ABGs, expiratory gas flow pattern, and clinical status (see APRV Protocol)

- APRV settings of P High-Low 35/0 and T High-Low 4.0/0.8 will yield a mean airway pressure of approximately 29 cms H_2O.

- Conventional modes of ventilation cannot maintain same mean airway pressure while limiting peak and/or plateau pressures to 35 cms H_2O and maintaining sufficient tidal ventilation.

Initial Set Up:

Activation in the Evita 2 Dura: Airway Pressure Release Ventilation (APRV)

1) Press the 'Other Modes' key

2) Turn dial knob to APRV

3) Set the ventilation pattern for APRV with the following parameters:

   a) Inspiratory pressure – P High
   b) Expiratory pressure – P Low
   c) Inspiratory time – T High
   d) Expiratory time T - Low
   e) O_2 concentration - O_2
   f) Pressure rise time - Slope

4) To set a specific parameter (a thru d), turn dial knob to respective parameter

5) Press dial knob (LED will turn yellow)

6) Turn dial knob to set value

7) Press dial knob to confirm value (LED will turn green)

8) To set a specific parameter (e thru f), press parameter screen key

9) Turn dial knob to set desired value

10) Press dial knob to confirm setting

11) Once all settings are confirmed toggle back to APRV and press the dial knob to activate mode of ventilation.

12) Press the Menu key to return to the main screen

13) If the ventilator is in Stand-by, depress Stand-by screen key to activate the mode

* Once APRV is activated any changes made to the mode of ventilation can be accessed by simply pressing the Settings menu key. This will prompt the user to the APRV menu.

Activation in the Evita 4: Airway Pressure Release Ventilation (APRV)

1) To activate, press Mode Settings

2) Touch the 'APRV" screen key (the LED will turn yellow)

3) Set the ventilation pattern for APRV with the following ventilation parameters:

    a) Inspiratory pressure – P High
    b) Expiratory pressure – P Low
    c) Inspiratory time - T High
    d) Expiratory time - T Low
    e) O_2 concentration - O_2
    f) Pressure rise time - Slope

4) To set a specific parameter, touch the respective screen knob

5) Turn dial knob to set value

6) Press dial knob to confirm value

7) Press the dial knob to activate mode of ventilation

8) Press the Mode Settings key to return to the main menu

*The modes setting screen can only hold 4 modes of ventilation. If APRV is NOT displayed, it can be activated in the following manner:

1) Touch the Configuration key

2) Touch the Ventilation screen key

3) Touch the corresponding numbers '3032'

4) Touch the Modes key

5) Touch the mode of ventilation you wish to remove from the original menu. You cannot remove a mode of ventilation that is in use. You must first change to another mode before it can be removed from the main menu.

6) Touch the mode to be removed from the main menu. A list will appear with every available mode the Evita 4 has to offer.

7) Turn dial knob to the corresponding mode to be placed in the main menu (APRV)

8) Press dial knob to confirm. APRV will now appear on the main menu of the Mode Settings screen.

Monitoring:

- ❑ Clinicians working with APRV monitor the same parameters used for any patient undergoing mechanical ventilation. In addition, the clinician must pay particular attention to the expiratory gas flow pattern (See: "APRV and Expiratory Gas Flow" document)

- ❑ Mean airway pressure has a linear relationship with oxygenation up to the point of alveolar over distension and creation of physiologic dead space

- ❑ Excessively high mean airway pressure may impair pulmonary perfusion, particularly in the presence of hypovolemia

- ❑ Exact range of mean airway pressure that can have significant reduction of cardiac output varies from patient to patient and is related to total thoracic compliance. Clinical correlation indicated.

- ❑ Low mean Paw is associated with alveolar collapse and shunting, and therefore, hypoxemia and increased pulmonary vascular resistance

- ❑ Expired minute volume - a result of the total of mandatory release volumes and patient spontaneous breaths - is typically lower during APRV than with

conventional modes of ventilation, yet with similar or improved alveolar ventilation.

- ❑ Release volume (the exhaled gas volume during the brief release periods), as with any pressure-targeted mode of ventilation, will vary with changes in compliance and resistance.

- ❑ Over time, increases in release volumes may represent an improvement in pulmonary compliance (recruitment of alveoli)

- ❑ Respiratory rate may vary and require assessment for pain, anxiety, delirium, improvement or deterioration of lung function

Weaning:

- ❑ The primary method to wean support in APRV is simultaneous manipulation of P High and T High. Technique known as 'Drop and Stretch' (See APRV Protocol).

    1) P High decreased by increments of 2-4 cm H_2O, and T High simultaneously lengthened in 0.5-2.0 second increments. If P High $\geq$35 cms H_2O, decrease P High by 2 cms H_2O and increase T High by 0.5 to 1.0 second. If P High $\leq$24 cms H_2O, decrease P High by 4 cms H_2O and increase T High by 2 seconds.

    2) Time interval between changes is patient dependent. Too slow, lengthens ICU stay. Too fast, promotes alveolar collapse.

    3) Goal is to arrive at CPAP 8-14 cm H_2O at which point the level of CPAP may be weaned further or the patient may be extubated from a 5-10 cm H_2O level.

    4) Prior to switching to CPAP, the P High is often 10-15 cm H_2O and the T High is approximately 10-15 seconds.

- ❑ During weaning the clinician closely monitors the mean Paw, SpO_2, exhaled minute volume and end tidal CO_2 level, as well as patient's overall comfort

- ❑ Incorporate ATC with institution of APRV. ATC reduces work of breathing imposed by artificial airways.

Frequently Asked Questions:

Q: Is there a good review article on APRV for the beginner or intermediate user of APRV?

A: Yes. Go to this page at the AACN.org website…
http://www.aacn.org/pdfLibra.NSF/Files/ci120205/$file/ci120205.pdf

Q: What is the maximum P High pressure used with APRV?
A: P High is typically set to a level equivalent to the plateau pressure in conventional modes. Patients recently intubated and APRV used as the first mode, commencing with a P High of 25-35 cms H_2O is common. Rarely is P High > 35 cms H_20 required. P High of 40-45 (or higher) may be indicated in isolated cases such as patients with decreased thoracic and/or abdominal wall compliance, e.g. obesity, chest restriction secondary to burns, restrictive dressings, chest wall edema, distended abdomens, or surgically packed abdomens. Extra pulmonary forces cause a decrease in the trans-pulmonary pressure for a given airway pressure.

Q: Should APRV be continued if oxygen saturation is not maintained, even with an FiO_2 of 1.0?
A: Although APRV is compatible with lung protective strategies and commonly utilizes lower airway pressures to produce similar oxygenation and ventilation compared to other conventional modes, there may be instances where APRV is not well tolerated. If APRV appears to be failing to meet ventilatory, oxygenation and lung protective goals, trialing another mode may be indicated. However, it is likely that other modes may also be poorly tolerated. Recruitment associated with APRV is occasionally seen immediately, though ultimate improvement of oxygenation is time-dependent. One study revealed maximal benefit after more than 8 hours with no further improvement after 16 hours (Sydow 1994).

Q: What P Low would you set if the PEEP was set at 15 cms H_2O on a conventional mode of ventilation?
A: P Low is set to zero cms H_2O independent of the previously set PEEP. End expiratory lung volume is maintained by adjusting the T Low. Expiratory flow rates are intentionally accelerated to allow the P High to be resumed as quickly as possible and facilitate secretion removal. A P Low set greater than zero increases expiratory resistance. Increased expiratory resistance slows gas flow, prolonging the time required for adequate ventilation and delays the onset of recruitment with P High.

Q: What T High and T Low would you use with APRV?
A: Initially, the P High is maintained for 4 – 6 seconds and the P Low is often set between 0.5 and 1 second, commonly 0.7 seconds. These times are respectively referred to as the T High and T Low. Precise T Low estimates may be derived from the expiratory gas flow graphic. T Low is set such that when the expiratory flow falls to approximately 75-25% of peak expiratory flow (depending upon disease process), P High is resumed. Limiting expiratory gas flow from falling to zero will minimize derecruitment by ensuring end expiratory lung volume is maintained. Generally, T Low will be shortened in restrictive diseases (ARDS) and lengthened in obstructive states (COPD and/or smaller endotracheal tubes). Adults may require T Lows as short as 0.3 seconds. Conversely, a T Low of 1.5 seconds may be indicated in patients with obstructive airway disease.

Q: What would you do if the respiratory rate was greater than 40/min (40-60) and the patient was on APRV?

A: Elevated respiratory rate (greater than 40 breaths/minute), may pose an unnecessary work of breathing. The first course of action is to evaluate for pain, anxiety, delirium increased neurogenic drive, worsening pulmonary pathology, severe metabolic acidosis, fever, or other metabolic load. Asses the ventilator and circuit to ensure settings are appropriate. Once these variables have been addressed, treated, or ruled out, there may be indication to increase P High and decrease T High to allow the machine to take an increased role in carbon dioxide removal.

Preservation of spontaneous breathing offers many advantages and therefore must be balanced against excessive work. Spontaneous breathing maintains tone in the diaphragm and provides protection against the compressive effects of the abdominal contents. These changes modify the pleural pressure and enhance regional ventilation by producing greater trans-pulmonary pressure. Therefore, recruitment can be accomplished without further increase in airway pressure. Additionally, spontaneous breathing facilitates complete assessment of the patient-ventilatory interaction, allowing improved clinical monitoring. Spontaneous breathing during APRV promotes coughing which enhances secretion clearance, reduces atelectasis, and ventilator associated pneumonia. Finally, by promoting comfort and spontaneous breathing, there will be less need for neuromuscular blocking (NMB) agents. In addition, complications of NMB agents and the associated cost are eliminated. Furthermore, rather than promoting muscle wasting, spontaneous breathing maintains muscular conditioning throughout the course of ventilation.

Q: If it is not possible to manually set a high or low PEEP alarm, why do high and low PEEP alarms sound with the Dräger ventilator?

A: The ventilator maintains and monitors PEEP. The 'PEEP valve inop!!!', 'Airway pressure low!!!' and 'PEEP High!!!' alarms are a built in safety feature with preset alarm points. They cannot be adjusted. The Dräger ventilator does not have a 'PEEP low' alarm. Activation criteria for 'PEEP valve inop!!!' is a measured PEEP level 5 cms H_2O or more below the set PEEP level for 10 consecutive breaths. The most common cause is a defective PEEP valve. 'Airway Pressure Low!!!' can be caused by a leak. Common reasons include a leaking endotracheal tube cuff, a leak in the circuit, or a disconnection.

The 'PEEP high!!!' alarm is activated when the PEEP measured at end expiration is elevated more than 5 cms H_20 above set PEEP. Three common causes include:

1) Expiratory system obstructed or increasing expiratory resistance.

Solution: Check entire expiratory limb of circuit and expiration valve. Look especially for a wet expiratory filter, kinked expiratory limb of the circuit, etc... Increased airway resistance can cause PEEP high alarm. Also, assess patient for wheezing, kinked or biting of the ET tube, and a narrow airway. Evaluate expiratory flow pattern for obstruction.

2) Faulty PEEP valve.
Solution: Call Dräger Service or hospital Bio-Medical services. A faulty PEEP valve is a technical problem and should be directed to Dräger Service.

3) Short release time or expiratory time. This problem may occur more readily with very short expiratory or release times, e.g. PC-IRV or APRV.
Solution: Resolution may be found with increases in the T Low or reduction in the inverse ratio of ventilation. The patient's condition must be considered when making such changes.

Comments:

- ❑ APRV settings of P High/Low 35/0 and T High/Low 4.0/0.8 will yield a mean airway pressure of approximately 29 cms H_2O. Conventional modes of ventilation cannot create similar mean airway pressure while limiting peak and/or plateau pressures to 35 cmH_2O and simultaneously maintaining sufficient tidal ventilation.
- ❑ End expiratory lung volume is maintained with time, i.e. short T Low
- ❑ PEEP is a fixed resistor and reduces expiratory flow rates
- ❑ Plateau pressure is used for recruitment (recruitment is an inspiratory phenomena). PEEP is used to prevent derecruitment (derecruitment is an expiratory phenomena).
- ❑ Typically P Low is set at 0 cms H_2O. An elevated P Low (like PEEP) acts as a fixed resistor throughout the entire release phase. Exhalation can be enhanced by elimination of resistance to flow. Expiratory flow and volume, without resistance, during short exhalation times may be superior to longer exhalation against resistance.
- ❑ Derecruitment is more likely with conventional PEEP than with the brief release of APRV, as the lung has a prolonged expiratory time (typical expiratory time in conventional PEEP ventilation>1.5 sec) to 'creep' towards the end expiratory pressure. Derecruitment occurs over a wide range of end expiratory pressure.
- ❑ It is a gross over-simplification to assume one PEEP level exists, above which all lung units are recruited and below which all lung units are derecruited

REFERENCES

Frawley PM. & Habashi, NM Airway pressure release ventilation: Theory and Practice. AACN Clinical Issues. 2001;12:234-246.
(http://www.aacn.org/pdfLibra.NSF/Files/ci120205/$file/ci120205.pdf)

Introduction

APRV is an established concept with original publications dating back 15 years (Stock, Downs, and Frolicher). The concept of high positive end expiratory pressure (PEEP) with intermittent releases dates back to the 70s (Douglas & Downs). Recent data suggests that previous ventilation standards may not be the optimal management for patients with acute lung injury (ALI). Researchers demonstrated tidal volumes of 12-15mls/kg, with little or no PEEP, adversely affect the lung by inducing both high- and low-volume lung injury. The ARDSNet study determined that patients with ALI and acute respiratory distress syndrome (ARDS) supported with "*mechanical ventilation with a lower tidal volume than is traditionally used results in decreased mortality and increased number of ventilator days without use (sic).*" (Brower, et al.). ARDSNet and other investigators, e.g. Amato, critically questioned traditional ventilator practice, though a comprehensive ventilatory strategy for the 21st century has not been forthcoming.

The evolving principles of new ventilatory strategies include:

1. Recognizing that a spectrum of different pressure-volume relationships exist within the same lung, making it impossible to ventilate all lung units within the lower and upper inflection points of a single pressure-volume curve.
2. Spontaneous breathing is beneficial by improving lung mechanics and V/Q mismatching.
3. Maintaining sufficient end-expiratory lung volume can prevent alveolar collapse (derecruitment).
4. Minimize oxygen toxicity and alveolar de-nitrogenation by using mean airway pressure to improve both oxygenation and alveolar stability.

The clinical challenges include the ability to support a spectrum of patients with ALI, limit ventilator associated lung injury (VALI) and facilitate spontaneous breathing. Preserving spontaneous breathing requires minimizing or eliminating ventilator dyssynchrony, providing a sufficiently long pressure period to maximize pulmonary interdependence and permit *pendeluft* (gas movement from alveoli to alveoli), and avoid derecruitment during exhalation without the untoward effects of high PEEP. Other important adjuncts include managing sedation to a level of comfort while preserving patient interaction; avoiding neuromuscular blocking agents; utilize prone positioning; and, retargeting ventilation goal with permissive hypercapnia.

Discussion

The airway pressure waveform of APRV appears similar to CPAP; however, during APRV the CPAP level is intermittently released at regular intervals (see Figure 1). These brief (typically less than one second) releases facilitate

tidal movement of gas, improving carbon dioxide clearance over CPAP. During APRV, the sustained positive airway pressure facilitates recruitment and oxygenation. Recruitment increases pulmonary surface area available for gas exchange, improving ventilation and lower minute ventilation requirements. An open breathing system as with Dräger Evita series ventilators enables APRV and permits spontaneous breathing to occur at any point in the respiratory cycle improving tolerance for sustained airway pressure.

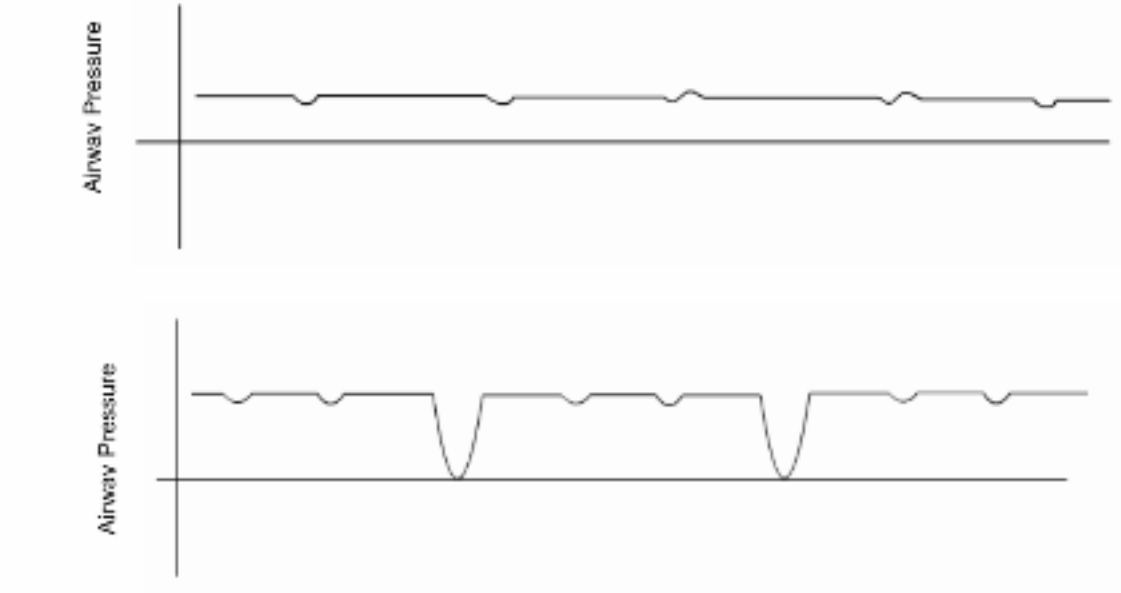

Figure 1: Diagram represents airway pressure changes in continuous positive airway pressure waveform (top) compared to airway pressure release ventilation waveform (bottom).

The advantages of spontaneous breathing include:

- Increased ventilation to poorly or non-ventilated lung regions, reducing the ventilation-perfusion mismatch

- Improved cardiac output secondary to improved venous return associated with decreased thoracic pressures during spontaneous inspiration. Spontaneous breathing decreases right atrial (P_{RA}) pressure, thereby increasing the gradient between the mean systemic venous pressure (P_{MS}) and the right atrium. An increased P_{MS} - P_{RA} gradient improves venous return and increases cardiac output.

- During spontaneous (unassisted) breathing, the posterior muscular sections of the diaphragm contracts, pulling the diaphragm caudad (downward) into the abdomen. Active diaphragm contractions lower pleural pressure surrounding the posterior segments of the lung, improving ventilation and minimizing posterior atelectasis. Diaphragmatic tone also counters thoracic encroachment of abdominal contents, minimizing the effects of reducing abdominal compliance.

Sustained Airway Pressure

Constant airway pressure may be advantageous for the:

- facilitation of alveolar recruitment
- enhancement of diffusion of gases (respiration)
- improving alveolar filling of units with slow time constants(from increased resistance)
- augmentation of "collateral channel" ventilation

Ventilation through collateral channels is limited in the healthy lung; however, in disease states associated with variable compliance and resistance, collateral ventilation may have an important role.

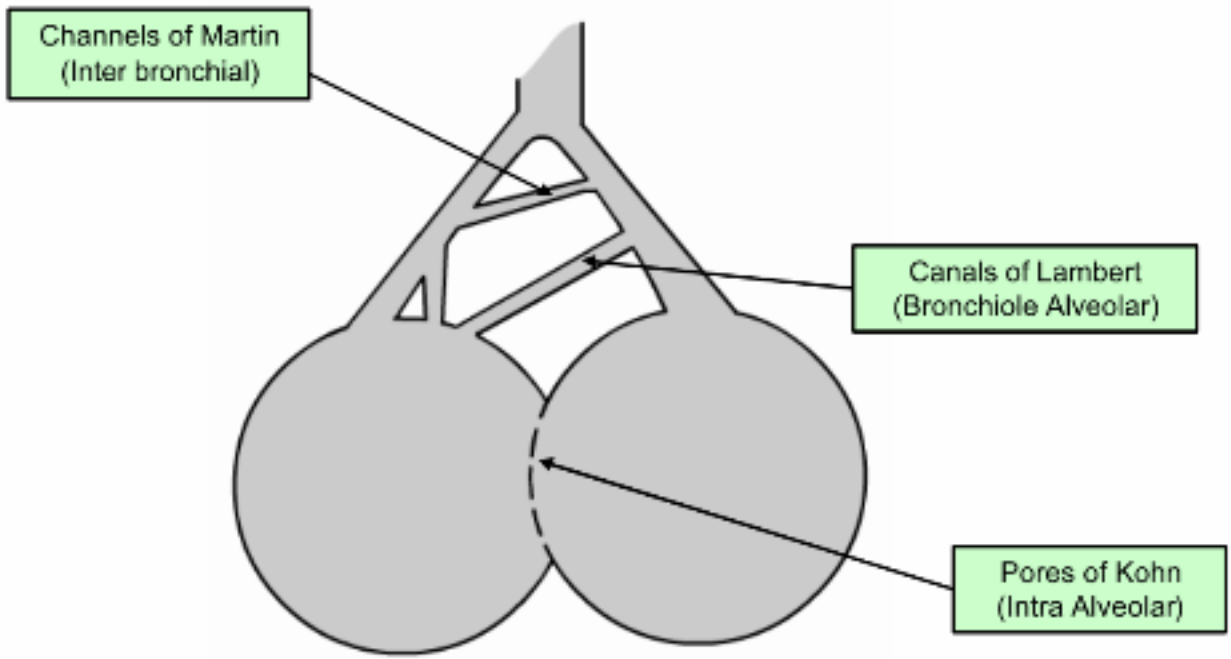

Figure 2: Collateral ventilation is time dependent. Longer inspiratory times may successfully ventilate adjacent alveoli which may not otherwise receive tidal flow via normal bronchial conduits due to long time constants or bronchial obstruction.

Collateral channels maintain alveolar ventilation through alternate pathways, despite reductions in ventilation through the major channels of ventilation. Ventilation through collateral channels depends upon maintenance of a constant airway pressure for relatively long periods of time and sustained lung volumes typically above functional residual capacity. Short inspiratory times will reduce the effectiveness of collateral ventilation. During APRV sustained pressure, may promote collateral ventilation to recruit alveoli, improving oxygenation and carbon dioxide clearance.

Release Volume

The brief, pressure release generates a volume known as the 'release volume'. Conceptually, release volumes may not equate to conventional tidal volumes. The primary difference is that the release volume is passive, secondary to the elastic recoil of the lung and thorax. The size of the release volume reflects the degree of recruitment sustained during the P High period and may include a portion of the inspiratory reserve volume (IRV). Release volumes may be large. While an exhaled volume of 1,100ml, for instance, would be excessive in conventional ventilation, it may represent an acceptable release volume in APRV, if significant recruitment is occurring.

$$\text{APRV Release Volume} = (x)\ \text{IRC} + (y)\ \text{FRC}_{\text{recruited}}$$

Where IRC is the inspiratory reserve capacity maintained with higher mean airway pressures and $\text{FRC}_{\text{recruited}}$ represents alveolar volume regained (recruited) by sustained airway pressure and limited release phase derecruitment. The factors "x" and "y" represent unknown amounts of each volume.

By comparison, during traditional ventilation, a pre-set tidal volume is forced into the lungs above a set or intrinsic PEEP level. Elevated airway pressures and tidal volumes may be key components of bio/baro/volutrauma. Depending upon the degree of lung distention and pathology, the inflated tidal volume may lead to alveolar overdistension and lung injury. Traditional exhaled tidal volumes represent the clinician-set volume and, less so, the degree of alveolar recruitment.

$$\text{Traditional Tidal Volume} = 6 \text{ to} 12 \text{mls/kg}$$

Patient Subsets

APRV has been utilized successfully in a variety of patient populations and appears particularly effective in patients with recruitable lung disease.

Applying APRV early in the disease process may prevent a derecruiting disease process from progressing to an unmanageable situation or limit the need for late application of rescue modes or levels of ventilation.

APRV has been successfully applied in a variety of situations and patient populations including, though not limited to:

- ❑ Adults (Rasanen, et al.)
- ❑ Pediatrics (Ryan Schultz, et al.)
- ❑ Cardio-thoracic (Garner, et al.)
- ❑ Trauma (Frawley & Cowan)
- ❑ Non-invasive ventilation (Jousela, et al.)
- ❑ Hemodynamically unstable patients (Falkenhain, et al.)

Initial APRV Settings

New technology requires an investment of time for clinicians to learn how to best incorporate technology into daily practice. APRV begins with the understanding of four parameters utilized to set and adjust the mode (see Figure 3).

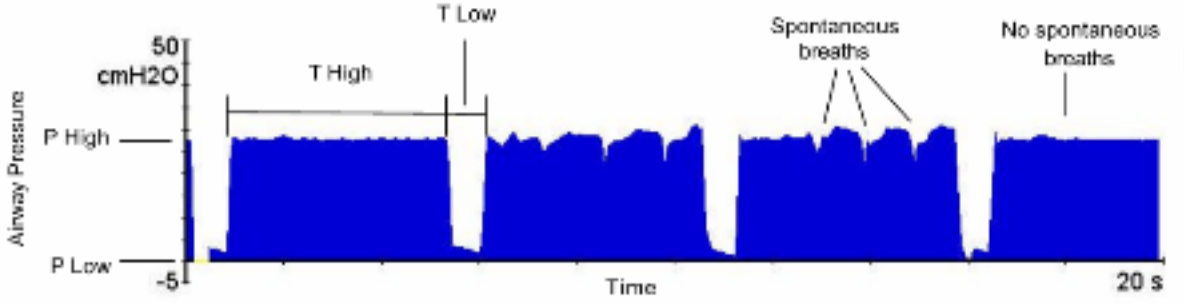

Figure 3: Terminology of APRV. P High is the higher of the two pressure levels. P Low results from the pressure release and is the lower of the two pressure settings. T high is the length of time for which P High is maintained. T Low is the length of time for which P Low is maintained.

Terminology

P High (high pressure) may be initially approximated by several options. One option is to set P High at a level approximating the plateau pressure of the prior conventional mode or as high as 35cms H_2O if the patient is newly intubated. An estimated initial setting of P High can be developed using the Oxygenation Index (OI) or using the mean airway pressure equation for APRV with certain assumptions (See APRV Protocol).

T High (time of high pressure duration) is initially set at 4-6 seconds. Extending the T High or CPAP period may be required to enhance CO_2 clearance (See ICON APRV protocol©).

P Low (low pressure) is set at 0 cms H_2O. Maintaining a P Low of 0 cms H_2O reduces downstream resistance during the release phase, increasing, expiratory flow rate. Expiratory flow rates are intentionally accelerated to accomplish carbon dioxide removal in the shortest period of time.

T Low (time of low pressure duration) manipulation requires an understanding of the gas flow waveform. The goal is to terminate the expiratory gas flow at about 75% to 25% of peak flow. Therefore, if peak expiratory gas flow reached 80L/min, termination would occur between 60 and 20 L/min. This is readily approximated by examining the expiratory gas flow waveform and reducing the T Low until flow ends within that zone (see Figure 4). The expiratory gas flow waveform is very useful in guiding adjustment of T Low for a variety of conditions, including low or high compliant lungs, normal airways or airways with obstructive airway diseases, or any combination of the above.

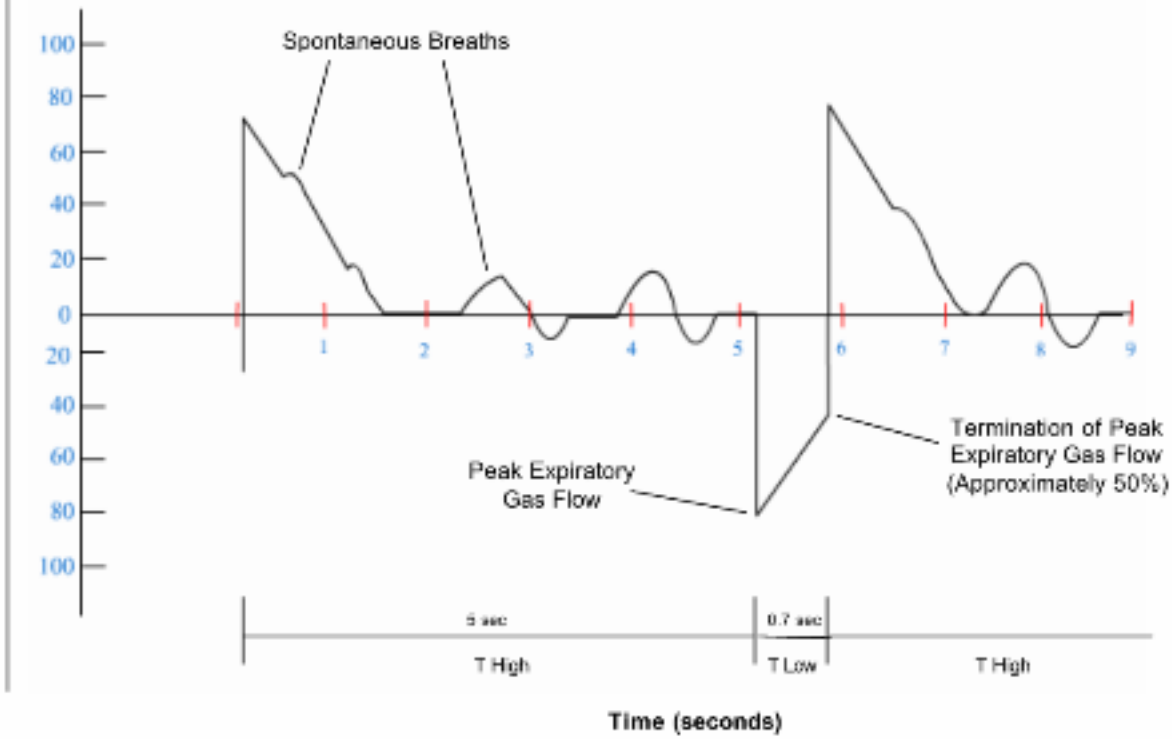

Figure 4: Inspiratory and expiratory gas flow pattern. Inspiration is above the horizontal axis, expiration below. T Low is adjusted such that expiration is truncated at 75 to 25% of peak expiratory flow.

Weaning

Weaning from APRV is accomplished by utilizing a strategy known as 'drop and stretch'. When the patient's status indicates reduction in ventilator support, the P High is dropped and the T High is stretched out sequentially. This process is continued until the P High is at a level of 10-15 cms H_2O and the T High is at 10-15 seconds. At this point, the patient is effectively on CPAP. With automatic tube compensation already activated, the patient is converted to CPAP. The conversion level to CPAP = P High minus three.

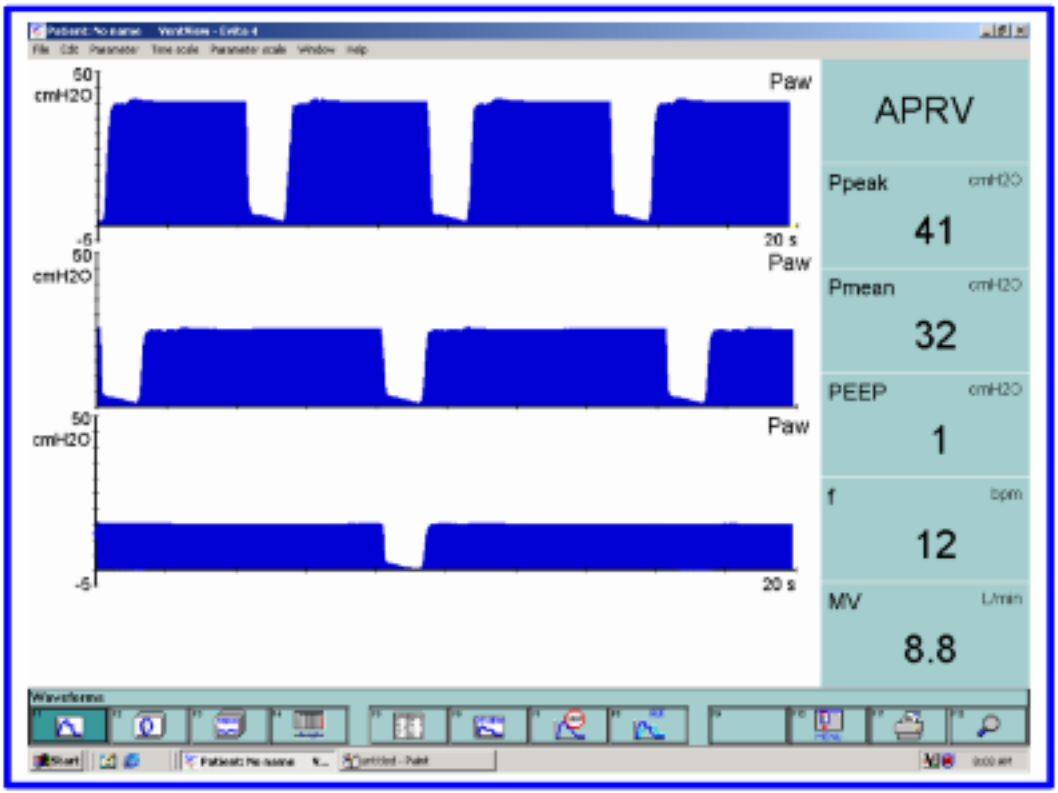

Figure 5: Diagrammatic representation of the 'Drop and Stretch' technique for weaning APRV. Upper tracing represents typical FULL support with APRV, the middle tracing represents an intermediate level of support and the lower tracing is approximating CPAP as the release periods become relatively rare.

Conclusion

Airway pressure release ventilation may provide a lung protective strategy, which limits both high- and low-volume lung injury. While the mode has been available in North America for over a decade (Dräger Medical), it is only recently that other ventilator companies have incorporated APRV.

Today, all major ventilator companies have included a mode that simulates APRV.

Few clinicians believe any single treatment or ventilator mode will be responsible for significantly reducing the mortality associated with ARDS. More likely, a combination of therapies will become the standard, including prone positioning, permissive hypercapnia, and APRV-like ventilatory strategies.

Selected References:

Frawley PM, Habashi NM. **Airway pressure release ventilation: Theory and practice.** *AACN Clinical Issues* 2001;12:234-246. **http://www.aacn.org/pdfLibra.NSF/Files/ci120205/$file/ci120205.pdf**

- ❑ Comprehensive review of theory and practice of APRV
- ❑ Detailed reference section
- ❑ Discusses initial settings, management, and weaning

Bottom Line:
Great place to start for both first time user and clinician with intermediate experience.

Garner W, Downs JB, Stock MC, Rasanen J. **Airway pressure release ventilation (APRV): a human trial.** *Chest* 1988;94:779-781.

- ❑ Early human study comparing APRV to conventional positive pressure ventilation (PPV)
- ❑ Examined 14 patients post-op following, cardio-thoracic surgery with subsequent mild acute lung injury
- ❑ APRV supported oxygenation and ventilation in patients with acute lung injury with much lower peak airway pressure than produced by conventional positive pressure ventilation

Bottom Line:
Optimal mode design will primarily provide CPAP as a primary intervention and secondarily augment alveolar ventilation.

Rasanen J, Cane RD, Downs JB, et al. **Airway pressure release ventilation during acute lung injury: a prospective multicenter trial.** *Crit Care Med.* 1991;19:1234-1241.

- ❑ Multicenter study (6 major referral institutions) comparing APRV to conventional ventilation
- ❑ Fifty patients with respiratory failure of multiple etiologies, including lung infection, sepsis, aspiration pneumonitis, pulmonary contusion, cardiogenic pulmonary edema, extrathoracic trauma and others
- ❑ APRV resulted in significantly lower $PaCO_2$ values despite lower average minute ventilation, implying decreased deadspace ventilation
- ❑ Three of the 50 patients were not adequately ventilated using APRV; these three failures was largely attributed to 'study design'

Bottom Line: In patients with mild to moderate ALI, APRV is an alternative to conventional ventilation for augmentation of alveolar ventilation.

Putensen C, Zech S, Wrigge H, et al. **Long-term effects of spontaneous breathing during ventilatory support in patients with acute lung injury.** *Am J Respir Crit Care Med* 2001;164:43-49.
http://ajrccm.atsjournals.org/

- ❑ A prospective, randomized study involving 30 multi-trauma patients
- ❑ Patients assigned to either spontaneous breathing with airway pressure release ventilation (APRV) or pressure-controlled (PC) for 72 hours
- ❑ Spontaneous breathing (APRV) was associated with increases in compliance, arterial oxygenation, cardiac index, oxygen delivery, reduced venous admixture and oxygen extraction
- ❑ In contrast, the PC group showed decreased compliance, arterial oxygenation, cardiac index, oxygen delivery, and higher requirement for sedation and vasoactive medications
- ❑ Spontaneous breathing (APRV) was also associated with statistically significant shorter duration of ventilatory support and shorter ICU stay

Bottom Line: Spontaneous breathing and maintenance of mean airway pressure improves pulmonary and hemodynamic function and is associated with significant reduction of ventilatory support and ICU stay.

Putensen C, Mutz NJ, Putensen-Himmer G, et al. **Spontaneous breathing during ventilatory support improves ventilation-perfusion distributions in patients with acute respiratory distress syndrome.** *Am J Respir Crit Care Med* 1999;159:1241-1248.
http://ajrccm.atsjournals.org/cgi/reprint/159/4/1241.pdf

- A prospective, randomized study involving 24 patients with acute respiratory distress syndrome (ARDS)
- Airway pressure release ventilation (APRV) with and without spontaneous breathing, or pressure support ventilation (PSV)
- Spontaneous breathing during APRV was associated with increased right ventricular end-diastolic volume, stroke volume, cardiac index, arterial oxygenation, oxygen delivery, and mixed venous oxygen tension, reduced pulmonary vascular resistance and oxygen extraction
- PSV was found not to be sufficient enough to counteract the V/Q mismatch caused by alveolar collapse

Bottom Line: Spontaneous breathing improves ventilation to well-perfused but under ventilated lung units decreasing shunt fraction. Ventilator support alone, without spontaneous breathing, increased dead-space ventilation by over ventilating under perfused regions.

Burchardi H. **New Strategies in mechanical ventilation for acute lung injury.** *Eur Respir J* 1996;9:1063-1072.

- A review article examining ventilatory strategies to optimize pulmonary mechanics
- The author reviewed small tidal volume ventilation, permissive hypercapnia, intrinsic PEEP, prolonged inspiratory times and spontaneous breathing
- When spontaneous breathing is possible, sedation and paralysis can be minimized thereby avoiding the complications of both medication groups
- To promote spontaneous breathing, newer generation ventilators are adapted to the patient, rather than adapting the patient to the ventilator by the use of analgesics, sedatives and/or paralytics. Employing sensitive demand valves are essential to this goal.
- "The most remarkable effect of maintained partially spontaneous breathing appears to be the improved recruitment of atelectatic and collapsed alveoli"

Bottom Line: Review of ventilatory strategies with support for the concept of spontaneous breathing.

Shultz T, Costarino Jr AT, Durning SM, Napoli LA, Schears G, Godinez RI, Priestley M, Dominguez T, Lin R, Helfaer M. **Airway pressure release ventilation in pediatrics.** *Pediatric Crit Care Med* 2001;2:243-246.

- Study was conducted in the intensive care unit at the Children's Hospital of Philadelphia and involved 15 children ages 1 to 15 years of age
- Each patient received both volume-controlled synchronized intermittent mechanical ventilation (SIMV) and APRV
- After baseline measurements were obtained, the patients were crossed over to the alternative study mode; (APRV to SIMV, or SIMV to APRV)
- Stabilization was again achieved and measurements repeated. After completion of the second study measurements, patients were placed on the ventilation modality preferred by the bedside clinician and was followed through weaning and extubation
- Clinicians at the bedside judged that:
  - 7 of the initial 9 'SIMV to APRV' patients should remain in APRV
  - 6 of the initial 6 'APRV to SIMV' patients should return to APRV
  - Ultimately, 13 out of 15 patients were managed and weaned to extubation using the APRV mode of ventilation
- Significant result were as follows:
  - Peak airway pressure (cms H_20) - SIMV 33 +/- 9; APRV 19 +/- 7
  - Plateau Pressure (cms H_20) - SIMV 23 +/- 8; APRV 18 +/- 6
  - Mean Airway Pressure (cms H_20) was similar at SIMV 11 +/- 4 vs. 10 +/- 4

Bottom Line: These findings suggest that APRV can provide adequate ventilation and oxygenation, yet greater patient comfort at lower airway pressures in children with moderate lung disease. Note: In this study, details of the APRV settings are not defined.

Jousela IT, Nikkio P, Tahvanainen J. **Airway pressure release ventilation by mask.** *Crit Care Med.* 1988;16:1250-51.

- A single case report
- APRV had been initiated during intubation
- Following extubation, APRV was continued
- There was essentially no difference in the ABGs when comparing APRV delivered via an endotracheal versus a face mask.
- In this case , APRV was developed by a combination of devices rather than a specific ventilator

Bottom Line: An early case study of non-invasive ventilation (NIV) applying APRV as a modality with good results. In this case, NIV permitted extubation without great risk.

Issue

Careful examination of the expiratory gas flow pattern can provide useful information for ventilator adjustment and define lung mechanics.

Evolution of expiratory gas flow patterns as a patient progresses through illness

The expiratory portion of the flow - time curve may provide valuable clinical information about the patient and the current ventilator settings. During APRV, exhalation can occur at any point within the respiratory cycle. Exhalation appears as a flow wave pattern below the horizontal axis of the flow - time curve. In APRV, exhalation can be divided into spontaneous exhalation and release phase exhalation In addition to the degree and duration of spontaneous exhalation, the characteristics of the release phase can be monitored and titrated to optimize mechanics and patient comfort.

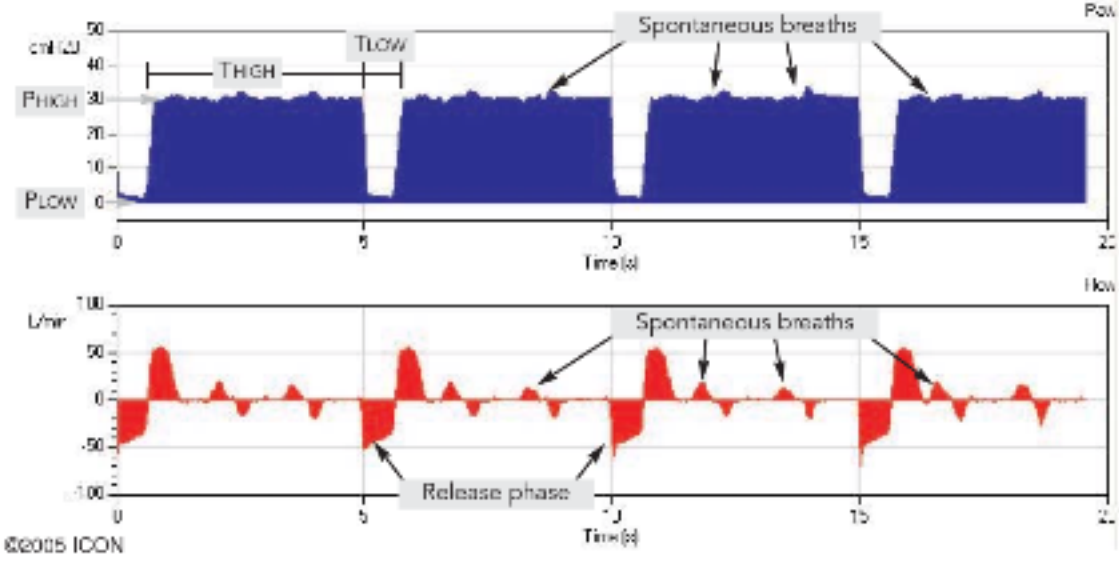

Figure 1: Typical Airway pressure and Flow waveforms of APRV. Note both the spontaneous efforts during the P high/T high period along with the release phase at the end of T high.

Adjusting T Low in the Presence of Changing Compliance

Lung derecruitment during exhalation is minimized by maintaining end expiratory lung volume. Measuring end expiratory lung volumes using intrinsic PEEP maneuvers with APRV will derecruit the lung and therefore should be avoided. Optimal end expiratory lung volume is difficult to calculate or measure, but may be optimized in APRV by terminating expiratory flow when flow has decreased to

between 75% and 25% of peak expiratory flow (Figure 2). The time to reach 75-25% of peak expiratory gas flow will vary depending upon multiple factors to include lung volume, time constants, airway size, and spontaneous breathing.

Ideal Expiratory Flow Pattern

If the goal is to terminate the expiratory gas flow at 50% of peak expiratory flow, the setting of the TLow will vary patient to patient according to that patient's compliance and resistance.

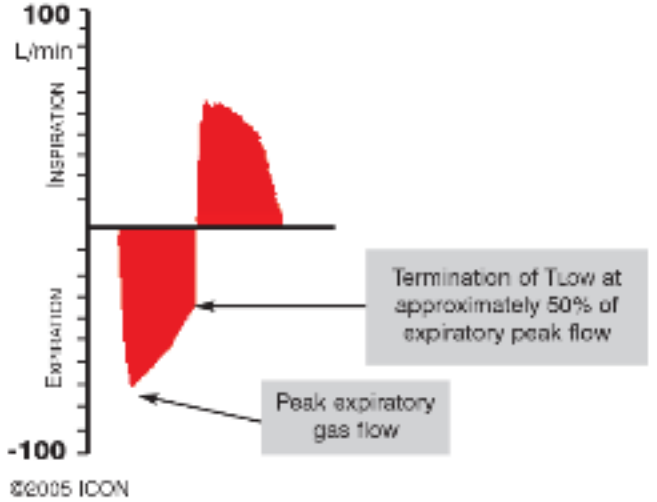

Figure 2: Typical expiratory flow curve during APRV. The release phase is represented below the horizontal axis. The flow pattern decelerates until the TLow is terminated. Therefore, termination of release phase flow is dependent on the release time. In this example, a TLow of 0.8 seconds yields a release phase termination at approximately 50% of the peak expirstory flow. Notice that the angle of deceleration is roughly 45 degrees.

Angle of Deceleration

The angle of deceleration (ADec) is the angle off of the Y axis that the expiratory gas flow travels during the release phase or TLow. A normal lung will have an ADec of approximately 45 degrees (Figure 3). As compliance worsens (ARDS), elastance will increase causing the ADec to decrease (Figure 4 and 5). In chronic obstructive pulmonary disease the elastance will decrease causing the ADec to increase (Figure 6).

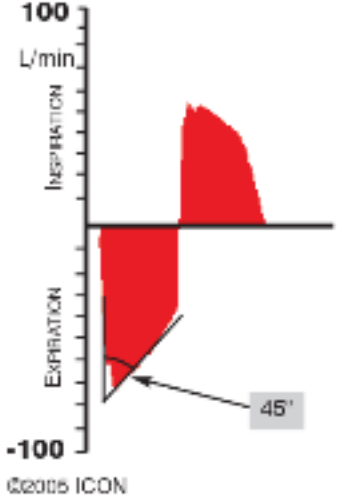

Figure 3: Normal angle of the time-flow curve is between 40° and 50°. TLow of 0.8 seconds terminates the expiratory limb at about 50% of its peak flow.

Restrictive: ***Not trapping enough***

If the lungs become less compliant, the release phase waveform shape will reflect increased recoil associated with acute restrictive lung/thoracic disease by decreasing the ADec. Due to high elastance (the inverse of compliance), a TLow of 0.8 seconds may now result in expiratory flow terminating at 10% of the peak expiratory flow.

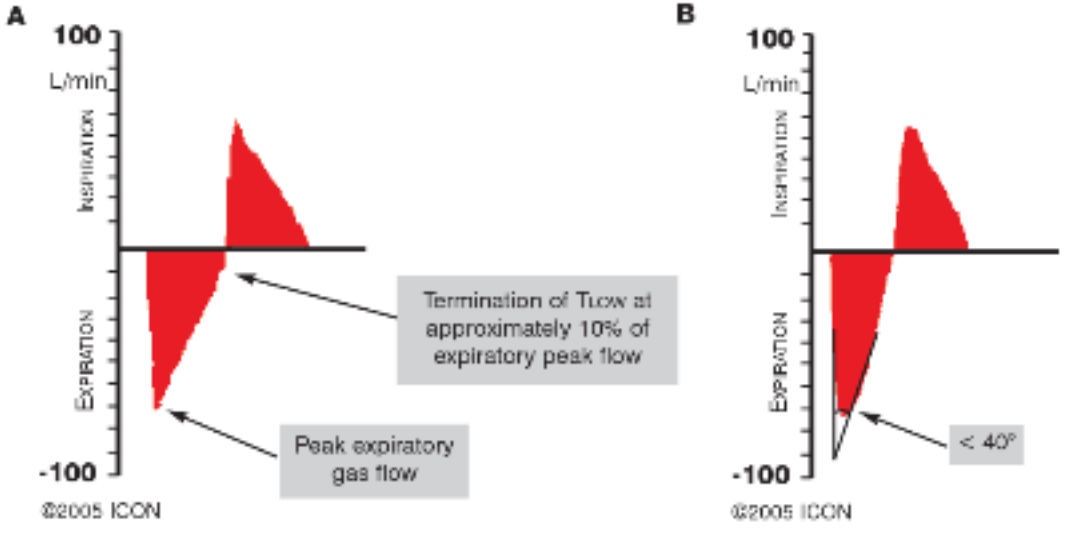

Figure 4: **A.** Release phase expiratory flow curve from a patient with low compliance (increased recoil) and a release time (T Low) of 0.8 seconds. Despite a brief release phase of 0.8 seconds, end expiratory flow termination point is reduced as a percentage of peak expiratory flow. In addition, as lung elastic recoil increases, the adec becomes steeper, **B.** Decrease in the angle of the expiratory flow curve associated with decreasing compliance.

To maintain end expiratory lung volume in the presence of increasing lung recoil, TLow should be shortened to reestablish termination of expiratory flow between 75 and 50% of peak flow. The actual amount TLow is shortened will be guided by the waveform. With the TLow set at 0.5 seconds, the waveform changes appearance (Figure 5). Maneuvers to increase alveolar recruitment, such as, increasing PHigh or THigh, or performing a defined "recruitment maneuver", may also improve the exhalation curve.

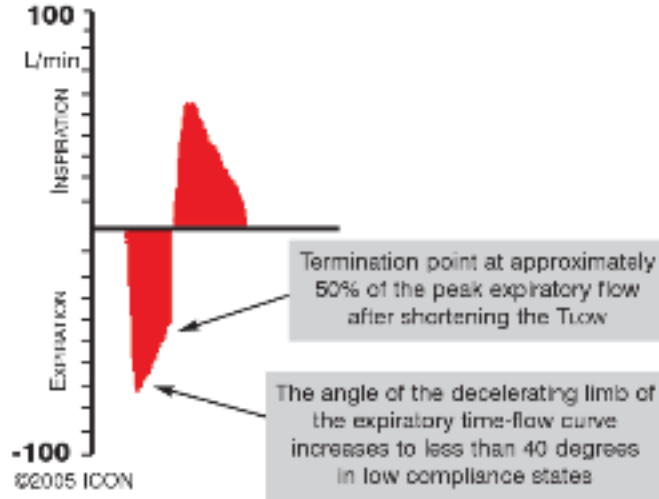

Figure 5: Release phase expiratory gas flow from a patient with low compliance (increased elastance) and a shortened TLow (0.5 seconds). TLow has been intentionally shortened to ensure that the terminal expiratory flow rate is about 75 to 25% of peak.

Obstructive Disease: *Increased Resistance*

In chronic obstructive pulmonary disease, airways lose supporting structure resulting in decreased elastance, airway obstruction and distal gas trapping. The collapsed airways function as a resistor to flow. Trapped gas creates intrinsic positive end expiratory pressure (PEEPi). Although intrinsic PEEP is recorded as one value, in reality, the recorded value reflects an average of many different regions, each with their own level of PEEP. In conventional ventilation, raising the extrinsic PEEP to a level at or near intrinsic PEEP opens more airways for a longer portion of the expiratory period. Extrinsic PEEP may allow for more complete exhalation and an ultimate reduction in intrinsic PEEP.

In the COPD patient being ventilated with APRV, collapsible airways are kept patent by utilizing a PHigh at or slightly above the patient's intrinsic PEEP level. This will offer the patient spontaneous breathing with minimal airway collapse. Automatic Tube Compensation may also be utilized to help unload the patient's work of breathing through an artificial airway. The angle of deceleration from the obstructed lung will be high (> 50 degrees) and may have a lower PEFR secondary to low elastance. In light of the high adec and low PEF, the termination point for the release flow should be between 50% and 25% of the PEF to allow for adequate CO_2 clearance. The ADec should also be evaluated for an upward truncation that reflects airway collapse. If this truncation is seen, release should end at the truncation point.

Often, in obstructive disease states, the ADec changes from normal to greater than 50 degrees. The ADec rises as the collapsed airways limit gas flow. Because this flow is "flattened" and is slower getting out, the expiratory flow is terminated between 50% and 25% of the PEF instead of 75% to 50% of the PEF. Achieving termination of expiratory gas flow at a point between 50 and 25% of peak flow usually requires setting a longer T*Low*. Expiratory times of 0.8 -1.2 seconds are typical, although times in excess of 2 seconds may be required in severe cases.

A rise in the ADec of the expiratory flow curve may also occur when a small artificial airway is used. Obstructions in the airways such as tumor, mucous plugs, secretions and or severe obstructive disease may also raise the ADec.

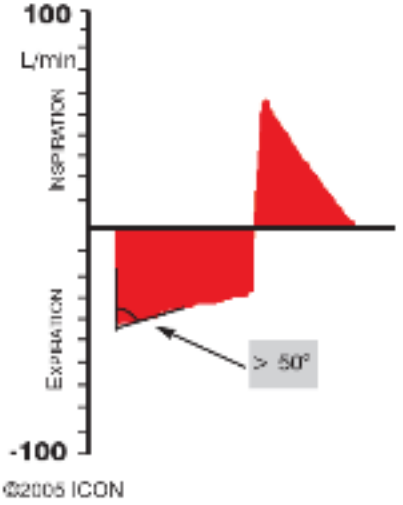

Figure 6: Flattened expiratory flow curve resulting from a small artificial airway limiting expiratory flow. T*Low* may need to be lengthened to compensate for the smaller tube.

Spike In Expiratory Flow Pattern

Initially, there may be a spike in the expiratory gas flow pattern. The expiratory spike represents an expulsion of compressed gas from within the ventilator circuit and is not coming from the patient. This false peak flow is not an accurate measure of true peak gas flow (Figure 7). The true peak flow begins where the spike ends. The value immediately following the spike should be used as the 100% point when calculating termination of T Low at 75 -25% of peak gas flow.

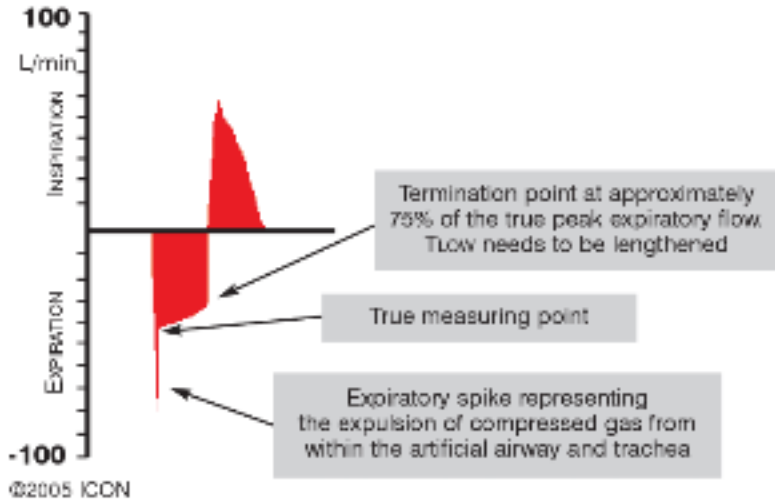

Figure 7: The initial spike represents the expulsion of compressed gas from within the ventilator circuit. The initial measuring point should be at the base of the spike, where true airway flow is seen

Screen Freeze Technique for Measuring Expiratory Flow

The Evita 4 and the Evita XL offer the ability to freeze the screen. This will assist in determining the percentage of trapped flow from the expiratory flow. Once the screen is frozen, choose an expiratory flow curve that reflects the average release pattern. Use the dial knob to move the cursor to the beginning of the peak expiratory flow rate (PEFR) and record the displayed flow (Remember not to include any circuit recoil in this measurement). Then, move the cursor to the end of expiration, calculate the termination point of expiratory flow rate (TP-EFR) and record the displayed flow. Now, divide TP-EFR by the PEFR and this will give you the fractional equivalent; multiply times 100 to get the percentage. If trapping 50%, the flow at the end of expiration will be half the flow displayed at the peak expiratory flow rate (Figure 8).

$$(\text{TP-EFR} - \text{PEFR}) \times 100 = \%\ \text{Trap}$$

where:

TP-EFR = Termination point of Expiratory Flow Rate
PEFR = Peak Expiratory Flow Rate

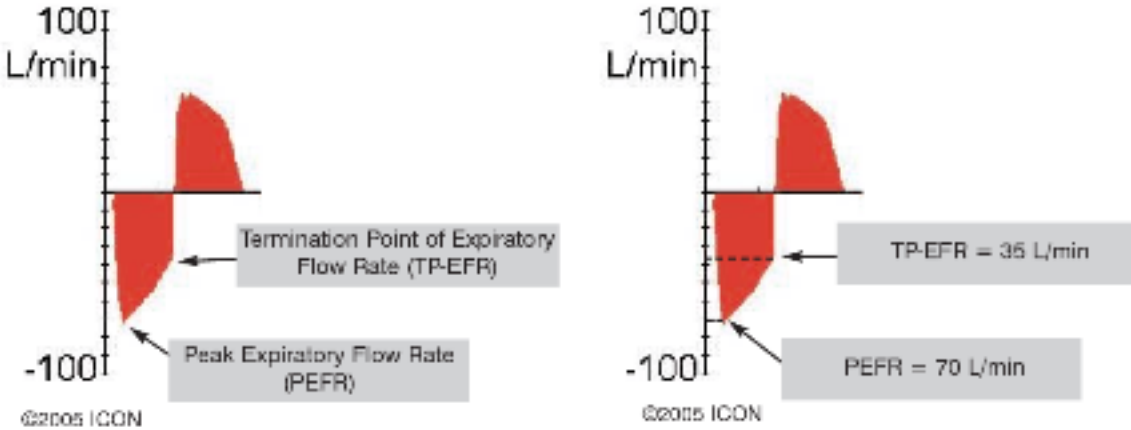

Figure 8: Using the freeze technique on the Evita 4 and the Evita XL to measure PEFR and expiratory flow rate termination point (TP-EFR). This example shows a PEFR of 70 L/min and a TP-EFR of 35 L/min; yielding a percent trapped of 50% (T-EFR / PEFR).

Summary

Utilization of the expiratory gas flow waveform is imperative for accurate titration of APRV parameters. Ascertaining the correct termination point on the expiratory gas flow waveform is crucial for proper pulmonary management. An unnecessarily short T*low* will be uncomfortable for the patient, limit exhalation and carbon dioxide removal, and may increase work of breathing. A T*low* set too long will promote derecruitment and worsening lung function and, ultimately, increase work of breathing. Of all APRV parameters to set appropriately, monitor and understand Tlow must be fully understood.

References

Habashi, N. Other approaches to open lung ventilation: airway pressure release ventilation. Crit Care Med 2005, 33(3): S228-240