APRV
Airway Pressure Release Ventilation

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Objectives

• What is APRV
• How to set it
• Why use it
• Benefits
• Controversies
What is APRV

- APRV was described more than 30 years ago (1987) by Stock and Downs as CPAP with intermittent release phase.
- APRV is classified as pressure controlled intermittent mandatory ventilation, and is typically applied using inverse inspiratory I:E ratios.
- There are both mandatory breaths (i.e. time-triggered and time-cycled), as well as spontaneous breaths (i.e. patient triggered and patient-cycled).
- Spontaneous breaths can occur both during and between mandatory breaths.
• P High: mandatory inspiratory pressure (Driving pressure)
• P Low: expiratory pressure (PEEP)
• T High: mandatory inspiratory time (I time)
• T Low: expiratory time
• Release rate: mandatory respiratory rate
<table>
<thead>
<tr>
<th><strong>P High</strong></th>
<th><strong>P Low</strong></th>
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<tbody>
<tr>
<td>P High is the applied pressure to airways</td>
<td>P Low is usually set at zero cmH2O</td>
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<tr>
<td>You want to use the least pressure possible to open the lung</td>
<td>Despite setting at zero, the pressure really never drops to zero given the very short T Low (release time) resulting in auto-PEEP</td>
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<td>Can set it according to plateau pressure in conventional ventilation</td>
<td>Can set below the upper inflection point of V-P curve</td>
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<tr>
<td>Can set according to end inspiratory transpulmonary pressure</td>
<td>Some add applied PEEP, how much ???????</td>
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P High & P Low
### T High
- The longer the time the better to sustain prolonged lung inflation
- Try to keep the lung 90-95% of the time inflated
- Try to minimize the release rate as possible 6-10/min if PH > 7.15 and PaCO2 are not too bad (Permissive hypercapnia)

### T Low
- The most important setting were exhalation and ventilation occurs
- Setting it as low as possible (0.2-0.6 seconds) to create auto-PEEP
- Can set according Peak Expiratory Flow to achieve 50-75%
- Can set to keep End expiratory (release) trans-pulmonary pressure > 0 cmH2O
Figure 2

T High & T Low

Inspiratory flow

Expiratory flow

PEFR (60 lpm)

50% of PEFR (30 lpm)

25%

75%

Full exhalation
How much Auto-PEEP are we creating
Is it enough to prevent lung collapse?
The answer: **NO IDEA**

Time Constant (TC) = Compliance × Resistance
Settings

- APRV is a form of “Open Lung Approach”: open the lung and keep it open
- Mean Airway Pressure (mPaw) is the name of the game in oxygenation
  \[(P \text{ High} \times T \text{ High}) + (P \text{ Low} \times T \text{ Low}) \over (T \text{ High} + T \text{ Low})\]
- Basically, you want the lowest P High, lowest P Low, Highest T High, lowest T Low
- You want the patient to breath spontaneously and contribute 25-30% of minute ventilation, less sedation
- Try NOT to add Pressure support above P High
Trouble Shooting

• PaO2 low:
  - increase T High
  - increase P High
  - decrease T Low
  - decrease sedation

• PaCO2 high/Low PH:
  - increase release rate
  - increase T Low
  - increase P High
  - decrease sedation to allow more spontaneous breaths
Confusion

- **Terminology:**
  different names of same mode in the literature
  
  Each ventilator company calls it something different
  
  APRV, BILEVEL, BIPAP, BIVENT, BIPHASIC, DUOPAP

- **Settings:**
  No agreed upon settings
  
  Some use low I:E ratio 1:1 – 2:1, other use > 10:1
  
  Some add P Low, some use 0
Benefits

- **Oxygenation:**
  Most studies have shown improved oxygenation compared to conventional modes with even lower pressure settings. Thus it has been described as a “rescue mode” in the difficult to oxygenate patient

- **Spontaneous Breathing:**
  Leads to improved ventilation-perfusion (V/Q) matching, decreased dead space through improvement in trans-pulmonary pressures in the juxtadiaphragmatic lung regions with resultant improved alveolar ventilation, and decreased atelectasis.
  Leads to improved diaphragmatic strength and decrease ventilator induced diaphragmatic injury and weakness
Benefits

• **Patient comfort:**

Patients have the ability to maintain their spontaneous breaths and tidal volumes with unrestricted flow pattern in relation to their metabolic needs which leads in better synchrony with the ventilator, improved work of breathing (WOB) despite less sedation and no neuromuscular blockade. Additionally patient’s have the ability to cough and clear their secretions which may translate to decrease VAP.

• **Hemodynamics:**

Multiple studies have documented improvements in venous return, Cardiac output, Oxygen delivery with less dosages of vasopressors and inotropes compared to conventional ventilation.

However other studies were not able to document such benefits
Long Inflation time

• A prolonged T high achieves the treatment goal of alveolar recruitment because it allows time for slow lung units in a non-homogenous lung field to inflate

• promote collateral ventilation through the channels of Martin, the pores of Kohn, and the canals of Lambert

• The longer the inflation time, the greater the lung areas potentially available for gas exchange

• The long inflation time creates higher mean airway pressure without the need for higher airway pressure or volume that can cause Ventilator Induced Lung Injury (VILI)

• [https://www.youtube.com/watch?v=oKH7CtsEgHw](https://www.youtube.com/watch?v=oKH7CtsEgHw)
• [https://www.youtube.com/watch?v=ZwzCRZcuTvY](https://www.youtube.com/watch?v=ZwzCRZcuTvY)
Lung Protective Effects

- Studies showed similar to superior safety of APRV compared to ARDS network low tidal volume
- As before, less inspiratory pressures used
- The long inflation time results in less frequent inflation and deflation which can contribute to shear stress on the alveoli (Atelectatotrauma)
- Some studies showed less development of ARDS in highly susceptible patients (shock, trauma) compared to conventional ventilation
Extra Vascular Lung Water
EVLW

We were able to document reduction in EVLW by switching from conventional ventilation to APRV.

The reduction of EVLW was correlated to the increased mPaw, and correlated to improved PaO2/FiO2, Mean Arterial Pressure, Cardiac output
Mortality?

- Older studies regarding mortality have been inconsistent regarding mortality some showing mortality but many not compared to conventional modes.
- Those studies are small and very heterogeneous regarding patient’s population, degree of sickness and the **settings used for APRV**.
- A recent well-done study in 2017 compared to low TV ARDS network settings in 138 patients showed superior ventilator free days, ICU days and Mortality.
- A new meta-analysis 2019 of 5 RCT of 330 patients showed improved hospital mortality at 28 days, ICU LOS, Ventilator free days, improved hemodynamics, no barotrauma.
Fear / Disadvantages

- High release volumes can cause Voluotrauma?
- Spontaneous breathing can cause worsening WOB and fatigue?
- Increased oxygen consumption by respiratory muscles?
- Short T Low and release rates can cause decreased minute ventilation resulting in worsening PaCO2 and PH?
- No large RCT showing mortality benefits
Unknowns

- How much Auto-PEEP are we creating using P Low of zero and very short T Low.
- Is it enough to prevent lung collapse and derecruitment?
- Does it really improve mortality?
- Can we agree on how to set it?
- Can we get it right?
References


• Habashi NM. Other approaches to open-lung ventilation: Airway pressure release ventilation. Crit Care Med. 2005;33:S228–40


• Daoud EG, HL Farag, RL Chatburn. Airway pressure release ventilation: what do we know? Respiratory care 2012; 57 (2), 282-292


References


- Early application of airway pressure release ventilation may reduce the duration of mechanical ventilation in acute respiratory distress syndrome


Thank You