

APRV

Airway Pressure Release Ventilation

Ehab Daoud MD, FACP, FCCP

Associate Professor of Medicine

JABSOM, University of Hawaii

Medical Director of respiratory Program, Kapiolani Community College

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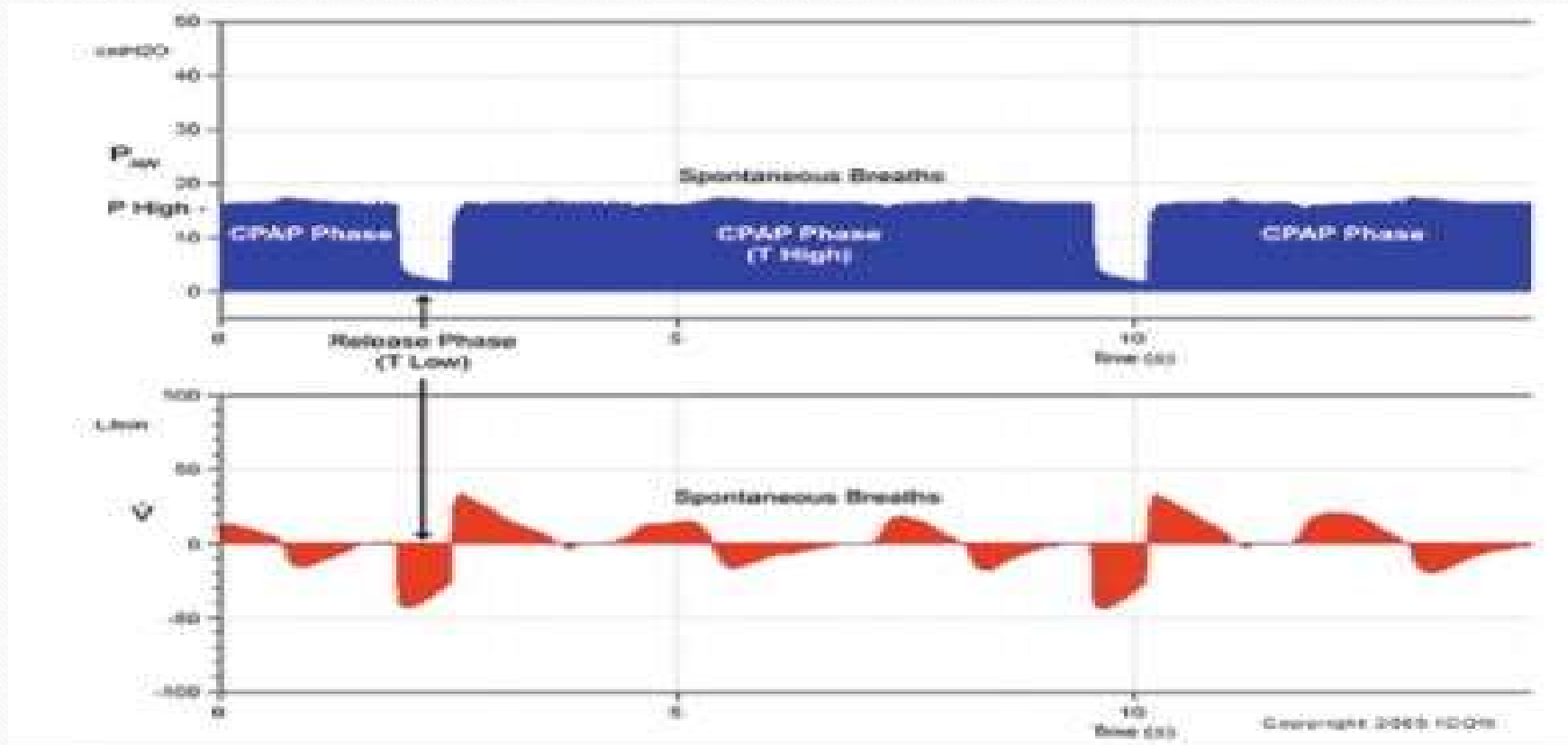


Objectives

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

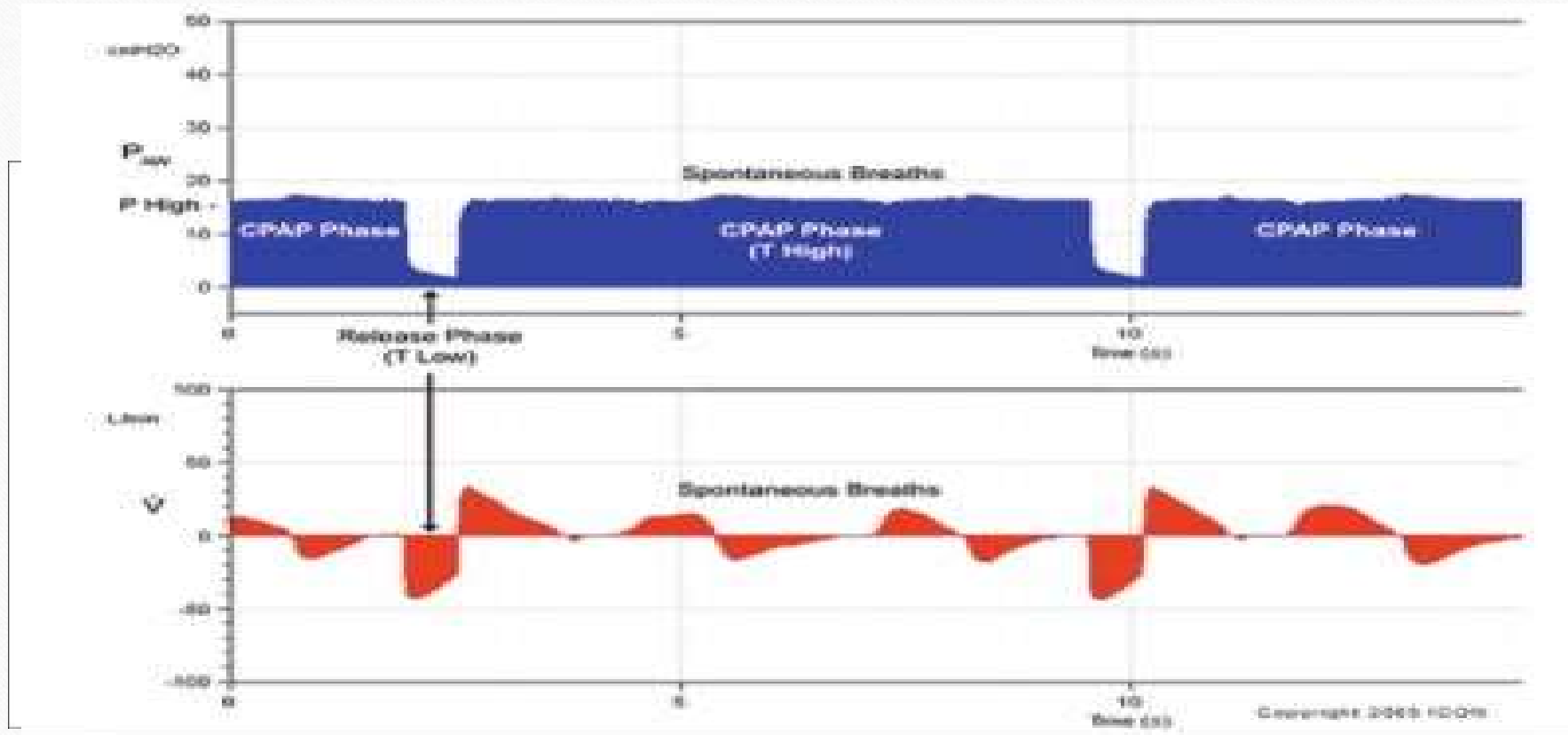
What is APRV

- 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



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Settings



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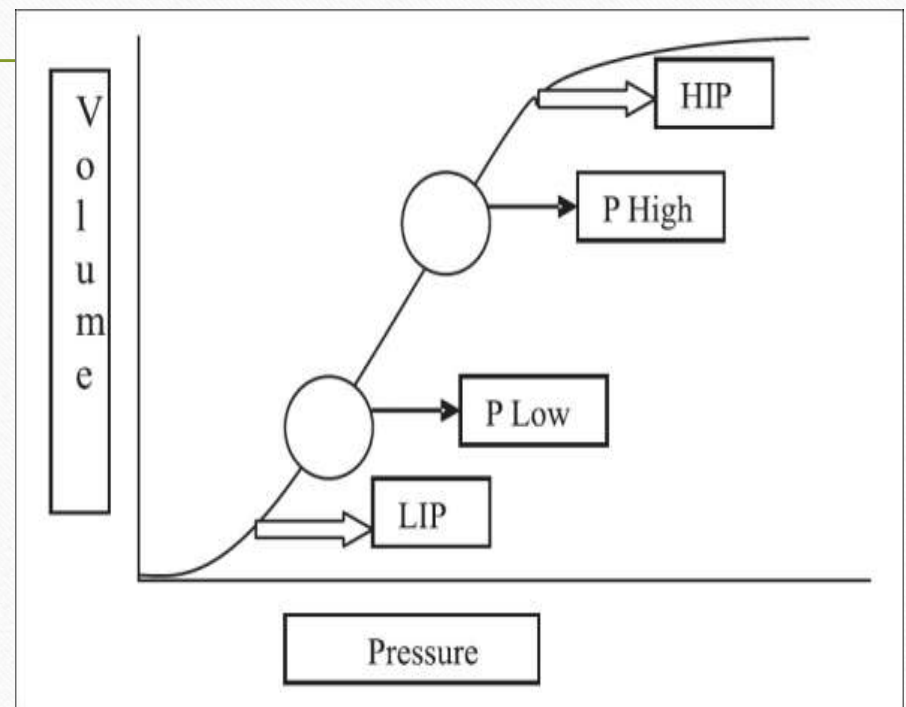
P High

- P High is the applied pressure to airways
- You want to use the least pressure possible to open the lung
- Can set it according to plateau pressure in conventional ventilation
- Can set below the upper inflection point of V-P curve
- Can set according to end inspiratory trans-pulmonary pressure

P Low

- P Low is usually set at zero cmH₂O
- Despite setting at zero, the pressure really never drops to zero given the very short T Low (release time) resulting in auto-PEEP
- Some add applied PEEP, how much ???????

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

T High & T Low

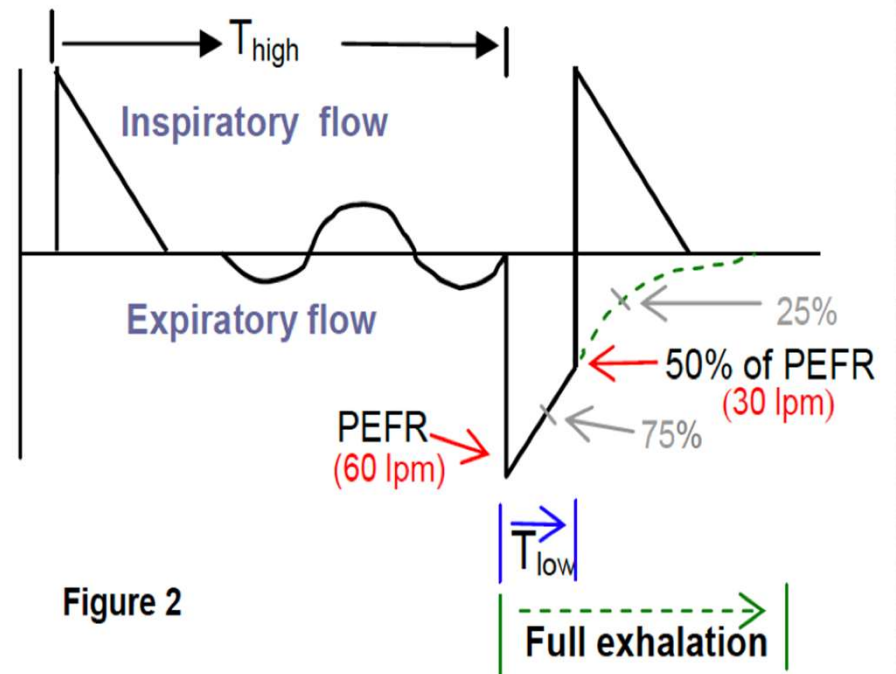
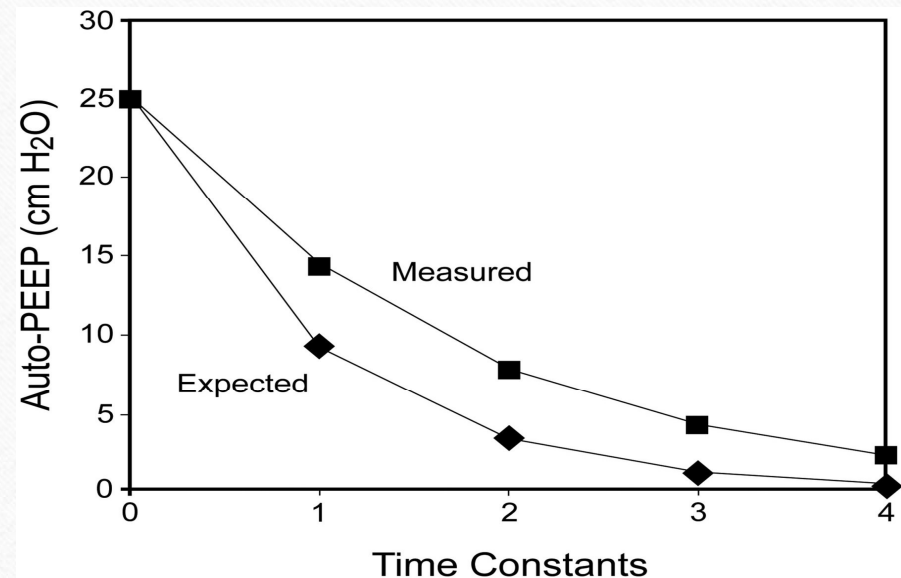
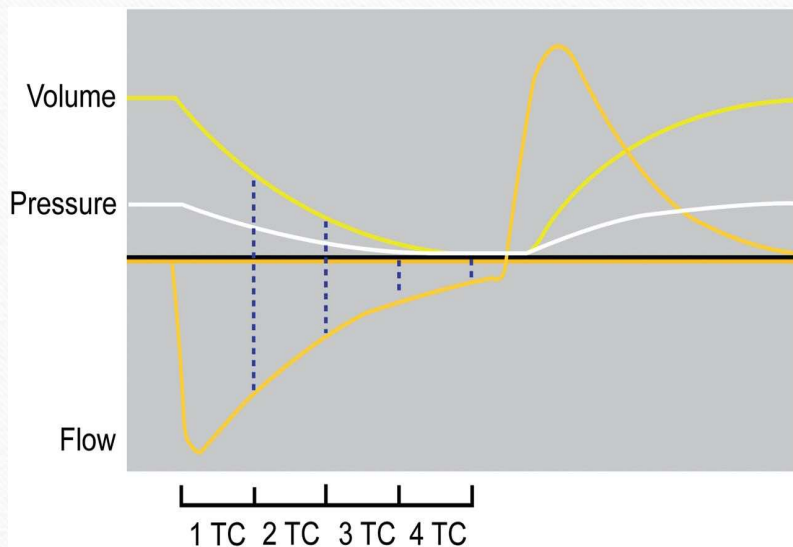


Figure 2

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



Time Constant (TC) = Compliance X 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

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

- 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 neuro-muscular blockade. Additionally patients 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=ZwzCRZcuTvY>

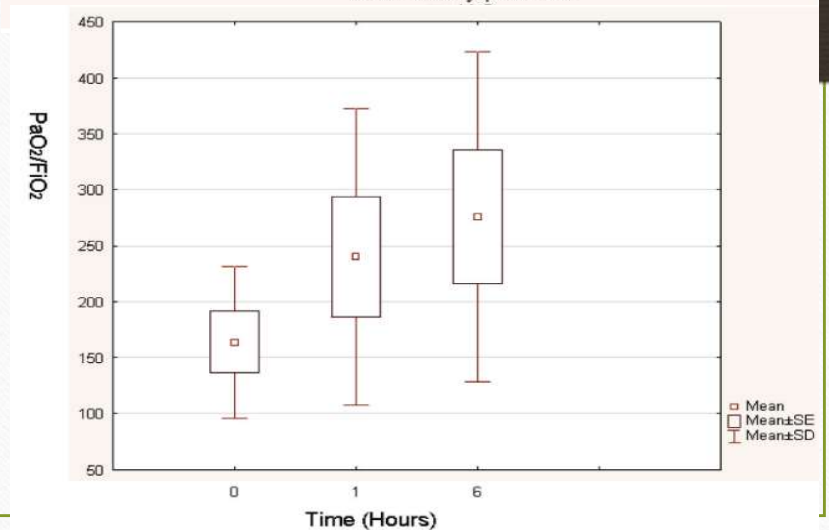
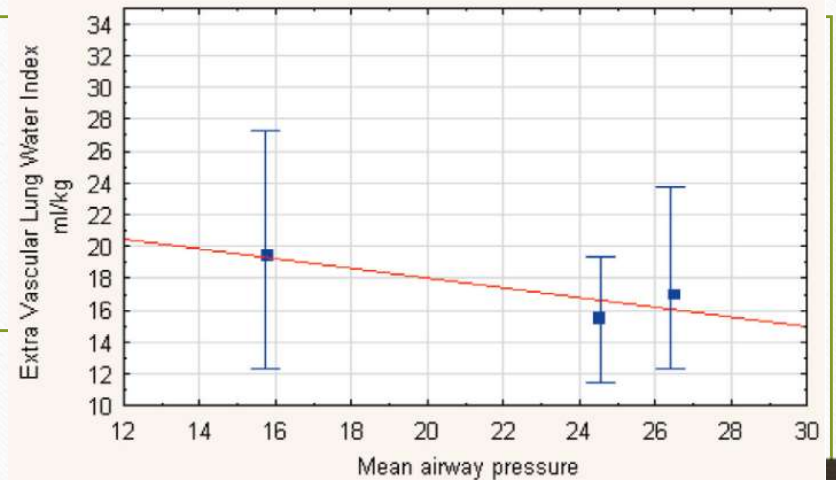
Lung Protective Effects

- 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.

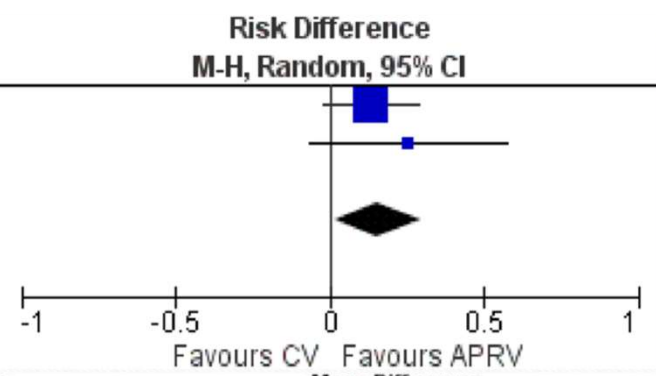
The reduction of EVLW was correlated to the increased mPaw, and correlated to improved PaO₂/FiO₂, Mean Arterial Pressure, Cardiac output



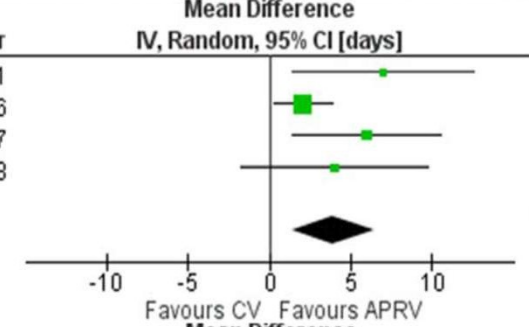
Mortality ?

- 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.

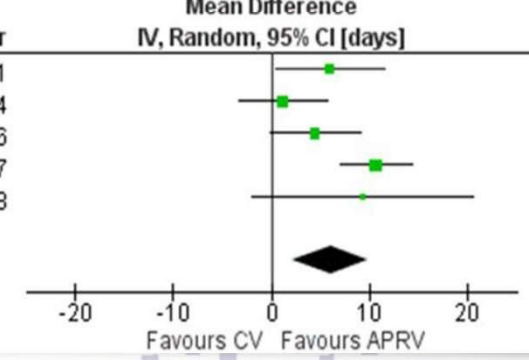
Study or Subgroup	CV		APRV		Weight	Risk Difference		Year
	Events	Total	Events	Total		M-H, Random, 95% CI	M-H, Random, 95% CI	
Zhou et al. 2017	25	67	17	71	81.5%	0.13	[-0.02, 0.29]	2017
Hirshberg et al. 2018	10	17	6	18	18.5%	0.25	[-0.06, 0.57]	2018
Total (95% CI)		84		89	100.0%	0.16	[0.02, 0.29]	
Total events	35		23					
Heterogeneity: Tau ² = 0.00; Chi ² = 0.45, df = 1 (P = 0.50); I ² = 0%								
Test for overall effect: Z = 2.22 (P = 0.03)								



Study or Subgroup	CV		APRV		Total	Weight	Mean Difference		Year
	Mean [days]	SD [days]	Mean [days]	SD [days]			IV, Random, 95% CI [days]	IV, Random, 95% CI [days]	
Putensen et al. 2001	30	7.75	15	23	15	15.5%	7.00	[1.45, 12.55]	2001
Li et al. 2016	9.5	3.2	26	7.4	26	49.3%	2.10	[0.33, 3.87]	2016
Zhou et al. 2017	20.67	16.67	67	14.67	71	20.4%	6.00	[1.40, 10.60]	2017
Hirshberg et al. 2018	10.5	11.24	17	6.47	18	14.8%	4.03	[-1.70, 9.76]	2018
Total (95% CI)			125		130	100.0%	3.94	[1.44, 6.45]	
Heterogeneity: Tau ² = 2.50; Chi ² = 4.75, df = 3 (P = 0.19); I ² = 37%									
Test for overall effect: Z = 3.08 (P = 0.002)									



Study or Subgroup	APRV		CV		Total	Weight	Mean Difference		Year
	Mean [days]	SD [days]	Mean [days]	SD [days]			IV, Random, 95% CI [days]	IV, Random, 95% CI [days]	
Putensen et al. 2001	13	7.75	15	7	15	19.9%	6.00	[0.45, 11.55]	2001
Varpula et al. 2004	13.4	9.31	30	12.2	28	23.2%	1.20	[-3.24, 5.64]	2004
Li et al. 2016	19.6	8.2	26	15.1	26	22.5%	4.50	[-0.15, 9.15]	2016
Zhou et al. 2017	16.33	10.59	71	5.67	67	25.6%	10.66	[6.99, 14.33]	2017
Hirshberg et al. 2018	15.33	19.31	18	6	17	8.8%	9.33	[-1.96, 20.62]	2018
Total (95% CI)			160		153	100.0%	6.04	[2.12, 9.96]	
Heterogeneity: Tau ² = 12.11; Chi ² = 11.35, df = 4 (P = 0.02); I ² = 65%									
Test for overall effect: Z = 3.02 (P = 0.003)									



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 ?

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Thank You

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