APRV APPLICATION

APRV – TCAV STRATEGY

Maria Madden MS, RRT-ACCS
VERO-Biotech/ICON-Clinical Specialist
AARC Chair – Adult Acute Section

DISCLOSURE

- Sponsored lectures and workshops
  - Intensive Care On-line Network (ICON)
  - Draeger

- VERO-Biotech Employee
- ICON Consultant

- None of the funding organizations or sponsors had any role in the design and conduct of any of the data presented;

OBJECTIVES

1. Review APRV settings and their rationale of the TCAV method
2. Review current literature supporting APRV-TCAV
3. Briefly discuss weaning from TCAV Method

ARDSnet
VA-AC

NIH NHLBI ARDS Clinical Network
Mechanical Ventilation Protocol Summary

INCLUSION CRITERIA: Acute onset of

1. $PaO_2/FiO_2 \leq 300$ (corrected for altitude)
2. Bilateral (patchy, diffuse, or homogeneous) infiltrates consistent with pulmonary edema
3. No clinical evidence of left atrial hypertension

PART I: VENTILATOR SETUP AND ADJUSTMENT

1. Calculate predicted body weight (PBW)
   Males = 50 + 2.3 [height (inches) - 60]
   Females = 45.5 + 2.3 [height (inches) -60]
2. Select any ventilator mode
3. Set ventilator settings to achieve initial V_T = 8 ml/kg PBW
4. Reduce V_T by 1 ml/kg at intervals ≤ 2 hours until V_T = 6ml/kg PBW.
5. Set initial rate to approximate baseline minute ventilation (not > 35 bpm).
6. Adjust V_T and RR to achieve pH and plateau pressure goals below.

OXYGENATION GOAL: PaO_2 55-80 mmHg or SpO_2 88-95%
Use a minimum PEEP of 5 cm H_2O. Consider use of incremental FiO_2/PEEP combinations such as shown below (not required) to achieve goal.

Lower PEEP/higher FiO2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12

FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

Higher PEEP/lower FiO2

FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5
PEEP	5	8	10	12	14	14	16	16

FiO_2	0.5	0.5-0.8	0.8	0.9	1.0	1.0
PEEP	18	20	22	22	22	24

PLATEAU PRESSURE GOAL: ≤ 30 cm H_2O
Check Pplat (0.5 second inspiratory pause), at least q 4h and after each change in PEEP or V_T.
If Pplat > 30 cm H_2O: decrease V_T by 1ml/kg steps (minimum = 4 ml/kg).
If Pplat < 25 cm H_2O and V_T< 6 ml/kg, increase V_T by 1 ml/kg until Pplat > 25 cm H_2O or V_T = 6 ml/kg.
If Pplat < 30 and breath stacking or dys-synchrony occurs: may increase V_T in 1ml/kg increments to 7 or 8 ml/kg if Pplat remains ≤ 30 cm H_2O.


THE SHOCK TRAUMA WAY
HABASHI METHOD
N94MD
TCAV

APRV

TCAV

Time Constant Adaptive Ventilation

Other approaches to open-lung ventilation: Airway pressure release ventilation

Nader M. Habashi, MD, FACP, FCCP

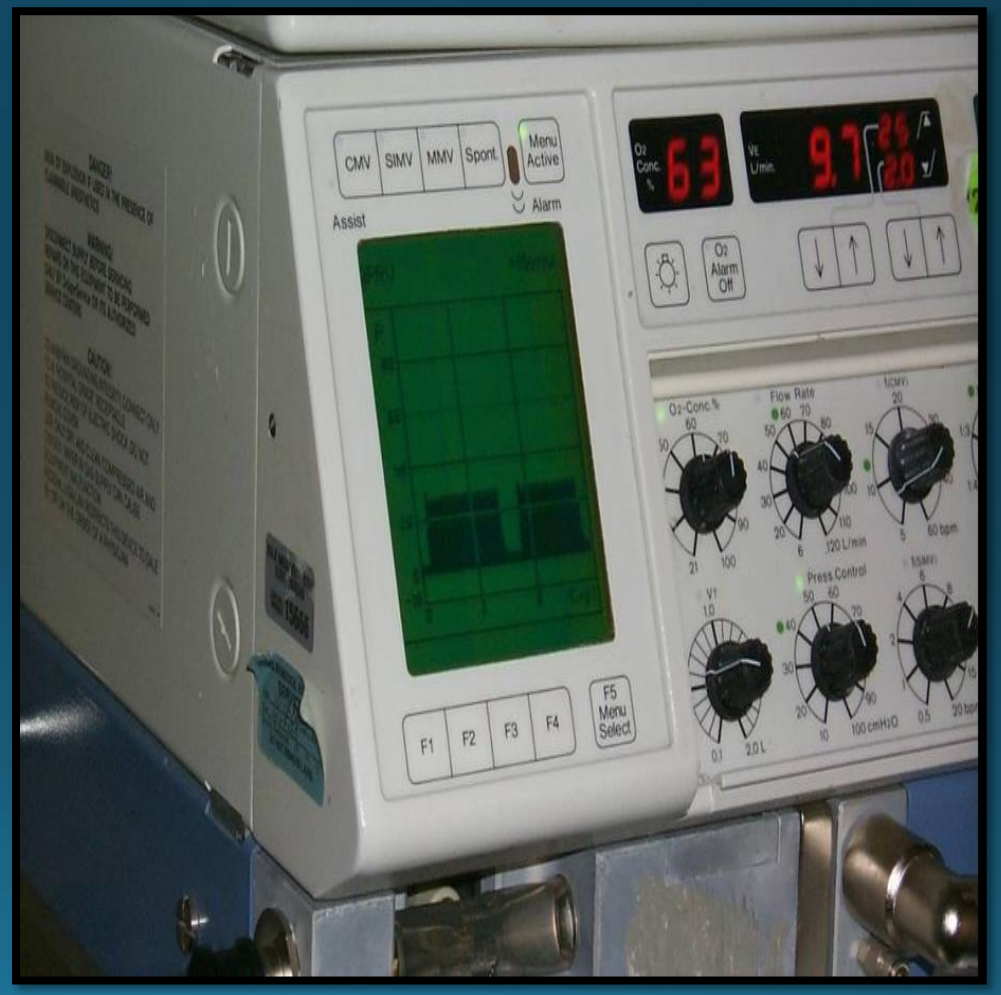
Crit Care Med 2005 Vol. 33, No. 3 (Suppl.)

Guidelines created after years of trying various APRV combinations.
T Low Updates since this article

Has anyone else written APRV guidelines?

APRV History

- APRV first described in 1987 by Dr. Stock and Dr. Downs
- APRV first made available on a commercial ventilator in 1987
- Some form of APRV is now available on almost all ICU ventilators
- APRV Guidelines developed by Dr. Habashi



APRV ALSO KNOWN AS ….

- APRV - Drager
- BiVent - Maquet
- BiLevel - Puritan Bennet
- APRV - Hamilton
- APRV/Biphasic - Viasys

EAST 2012 PLENARY PAPER

Early stabilizing alveolar ventilation prevents acute respiratory distress syndrome: A novel timing-based ventilatory intervention to avert lung injury

Shreyas Roy, MD, CM, Benjamin Sadowitz, MD, Penny Andrews, RN, Louis A. Gatto, PhD, William Marx, DO, Lin Ge, PhD, Guirong Wang, PhD, Xin Lin, PhD, David A. Dean, PhD, Michael Kuhn, BA, Auyon Ghosh, BSc, Joshua Satalin, BA, Kathy Snyder, BA, Yoram Vodovotz, PhD, Gary Nieman, BA, *and* **Nader Habashi, MD,** *Syracuse, New York*

EARLY AIRWAY PRESSURE RELEASE VENTILATION PREVENTS ARDS—A NOVEL PREVENTIVE APPROACH TO LUNG INJURY

Shreyas Roy,* Nader Habashi,† Benjamin Sadowitz,* Penny Andrews,† Lin Ge,* Guirong Wang,* Preyas Roy,‡ Auyon Ghosh,* Michael Kuhn,§ Joshua Satalin,* Louis A. Gatto,‖ Xin Lin,¶ David A. Dean,¶ Yoram Vodovotz, and Gary Nieman***

**Cardiopulmonary and Critical Care Laboratory, Department of Surgery, Upstate Medical University, Syracuse, New York; †Multitrauma Unit, R. Adams Cowley Shock Trauma Center, Baltimore, Maryland; ‡University of Chicago, Chicago, Illinois; §Department of Nutrition, Columbia University, New York; ‖Department of Biology, SUNY Cortland, Cortland; ¶Department of Pediatrics, Neonatology University of Rochester Medical Center, Rochester, New York; and **Department of Surgery, University of Pittsburgh, Pittsburgh, Pennsylvania*

Shock 2013 39(1):28-38

Published in final edited form as:
Shock. 2013 September ; 40(3): 210–216. doi:10.1097/SHK.0b013e31829efb06.

Preemptive Application of Airway Pressure Release Ventilation (APRV) Prevents Development of Acute Respiratory Distress Syndrome (ARDS) in a Rat Traumatic Hemorrhagic Shock Model

Shreyas K. Roy, M.D., C.M.[1], Bryanna Emr, M.D.[1], Benjamin Sadowitz, M.D.[1], Louis A. Gatto, Ph.D.[1,2], Auyon Ghosh, B.Sc.[1], Joshua M. Satalin, B.S.[1], Kathy P. Snyder, B.S.[1], Lin Ge, Ph.D.[1], Guirong Wang, Ph.D.[1], William Marx, D.O.[3], David Dean, Ph.D.[4], Penny Andrews, R.N.[5], Anil Singh, M.D.[1], Thomas Scalea, M.D.[5], Nader Habashi, M.D.[5], and Gary F. Nieman, B.A.[1]

APRV-TCAV when applied early reduces the pulmonary edema with a constant airway pressure (P High) for > 90% of the duration of the breath (T High)

Keeping the lungs open, minimizing atelectasis leads to decreased inflammatory mediators from being released

SUNY UPSTATE

- Highly translational 48 hour porcine model with ~40 kg pigs
- Sepsis, ischemia/reperfusion injury
- Animals were all ventilated with conventional ventilation during the operative procedure
- For subsequent 48 hours, transitioned to either non-protective ventilation [NPV] (10 ml/kg and PEEP 5), ARDSnet (low tidal volume strategy) or APRV
- All APRV animals maintained a PF ratio >300 throughout the entire experiment while on FiO_2 21% while all in the NPV and ARDSnet groups developed ARDS within 48 hours

ORIGINAL ARTICLE

Early application of airway pressure release ventilation may reduce mortality in high-risk trauma patients: A systematic review of observational trauma ARDS literature

Penny L. Andrews, RN, BSN, Joseph R. Shiber, MD, Ewa Jaruga-Killeen, PhD, Shreyas Roy, MD, CM, Benjamin Sadowitz, MD, Robert V. O'Toole, Louis A. Gatto, PhD, Gary F. Nieman, BA, Thomas Scalea, MD, *and* **Nader M. Habashi, MD,** *Baltimore, Maryland*

BACKGROUND: Adult respiratory distress syndrome is often refractory to treatment and develops after entering the health care system. This suggests an opportunity to prevent this syndrome before it develops. The objective of this study was to demonstrate that early application of airway pressure release ventilation in high-risk trauma patients reduces hospital mortality as compared with similarly injured patients on conventional ventilation.

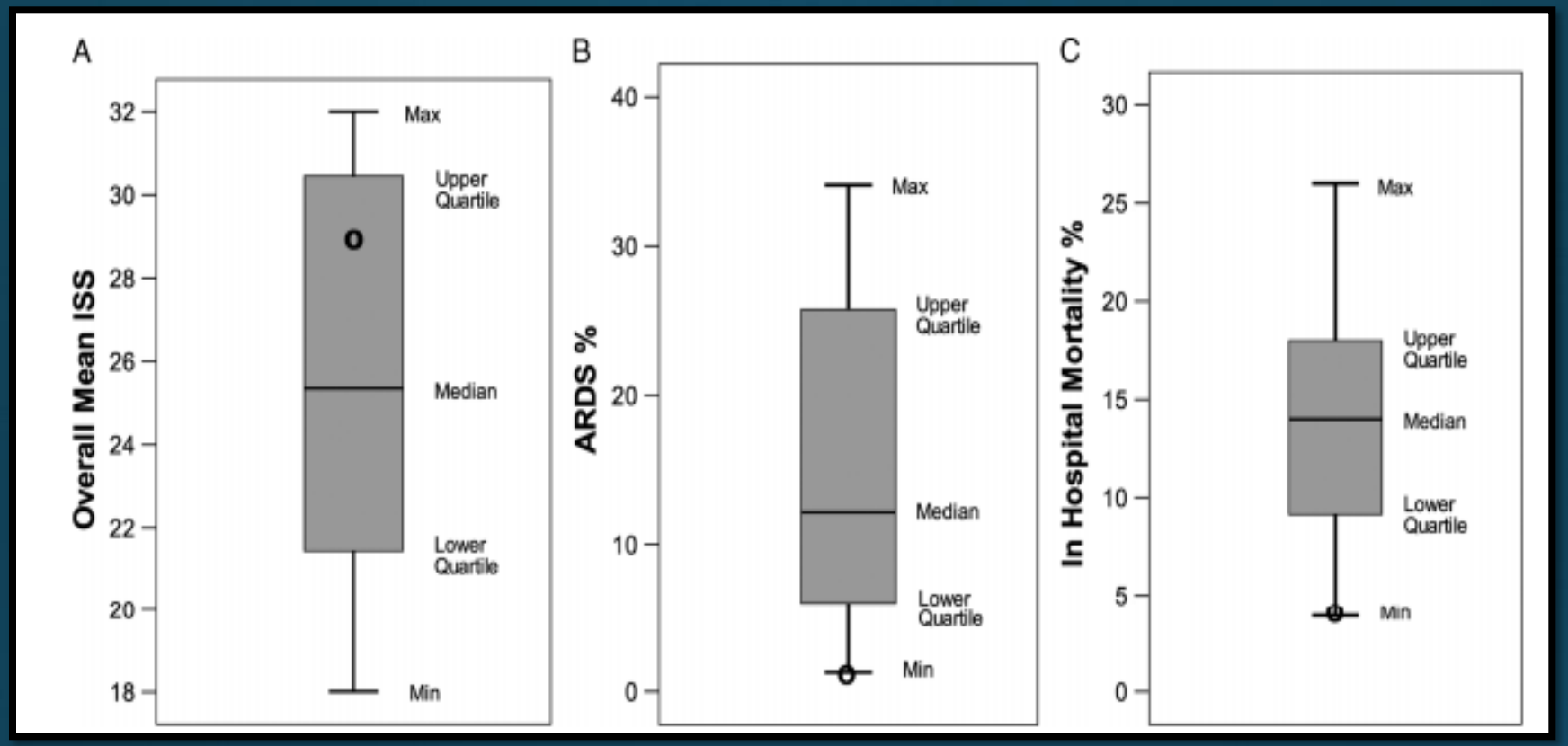
METHODS: Systematic review of observational data in patients who received conventional ventilation in other trauma centers were compared with patients treated with early airway pressure release ventilation in our trauma center. Relevant studies were identified in a PubMed and MEDLINE search from 1995 to 2012 and included prospective and retrospective observational and cohort studies enrolling 100 or more adult trauma patients with reported adult respiratory distress syndrome incidence and mortality data.

RESULTS: Early airway pressure release ventilation as compared with the other trauma centers represented lower mean adult respiratory distress syndrome incidence (14.0% vs. 1.3%) and in-hospital mortality (14.1% vs. 3.9%).

CONCLUSION: These data suggest that early airway pressure release ventilation may prevent progression of acute lung injury in high-risk trauma patients, reducing trauma-related adult respiratory distress syndrome mortality. (*J Trauma Acute Care Surg*. 2013;75: 635–641.

LEVEL OF EVIDENCE: Systematic review, level IV.

KEY WORDS: Airway pressure release ventilation; APRV; ARDS; adult respiratory distress syndrome; ALI.



CENTER	ISS	INCIDENCE %	MORTALITY %
STC ARDS	28.5	1.4	3.9
NATIONAL ARDS	26.2	13.5	14.2

J Trauma Acute Care Surg Volume 75, Number 4

ORIGINAL

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

Yongfang Zhou, Xiaodong Jin, Yinxia Lv, Peng Wang, Yunqing Yang, Guopeng Liang, Bo Wang and Yan Kang*

- 138 patients with ARDS who received mechanical ventilation for <48 h
- Patients were randomly assigned to receive APRV (n = 71) or LTV (n = 67).

Day 3 of enrollment	APRV	LTV
P/F RATIO	280	180
$PaCO_2$	40.8	42.3
PaO_2	116.2	84.8

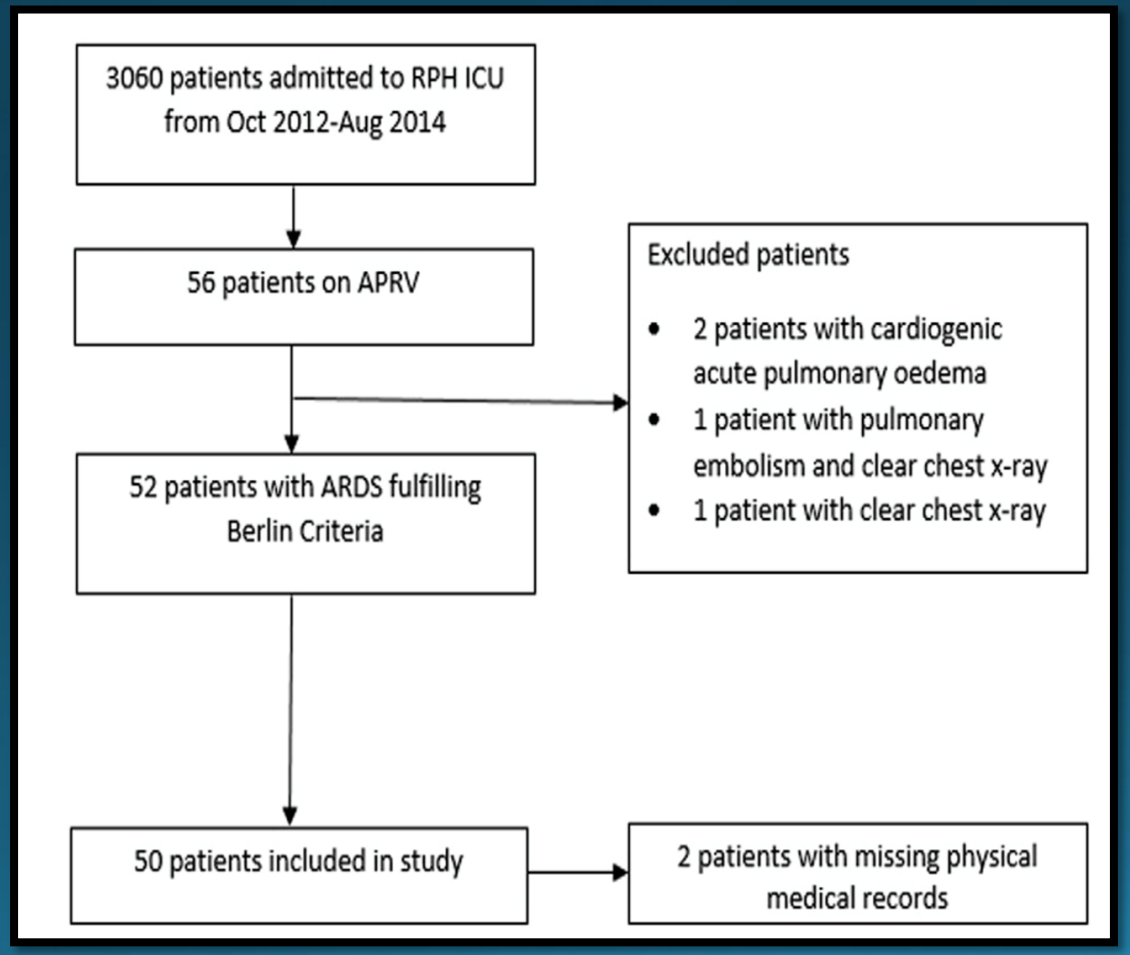
Intensive Care Med (2017) 43:1648–1659
DOI 10.1007/s00134-017-4912-z

Journal of Critical Care
Volume 34, August 2016, Pages 154-159

Outcomes/Predictions

Characteristics and outcomes of patients treated with airway pressure release ventilation for acute respiratory distress syndrome: A retrospective observational study ☆

Jolene Lim MBBS (Hon) [a, 1], Edward Litton MB, ChB, MSs, FCICM [b, c], Hayley Robinson BMedSci (Hon), MBBS (Hon) [d], Mike Das Gupta [e, 2]



ELSEVIER

Journal of Critical Care

Volume 34, August 2016, Pages 154-159

Outcomes/Predictions

Characteristics and outcomes of patients treated with airway pressure release ventilation for acute respiratory distress syndrome: A retrospective observational study ☆

Jolene Lim MBBS (Hon) [a, 1], Edward Litton MB, ChB, MSs, FCICM [b, c], Hayley Robinson BMedSci (Hon), MBBS (Hon) [d], Mike Das Gupta [e, 2]

50 patients placed on APRV

- 34 met ECMO criteria based on CESAR study
- 1 required ECMO

Why aren't we using APRV-TCAV before V V ECMO for ARDS?

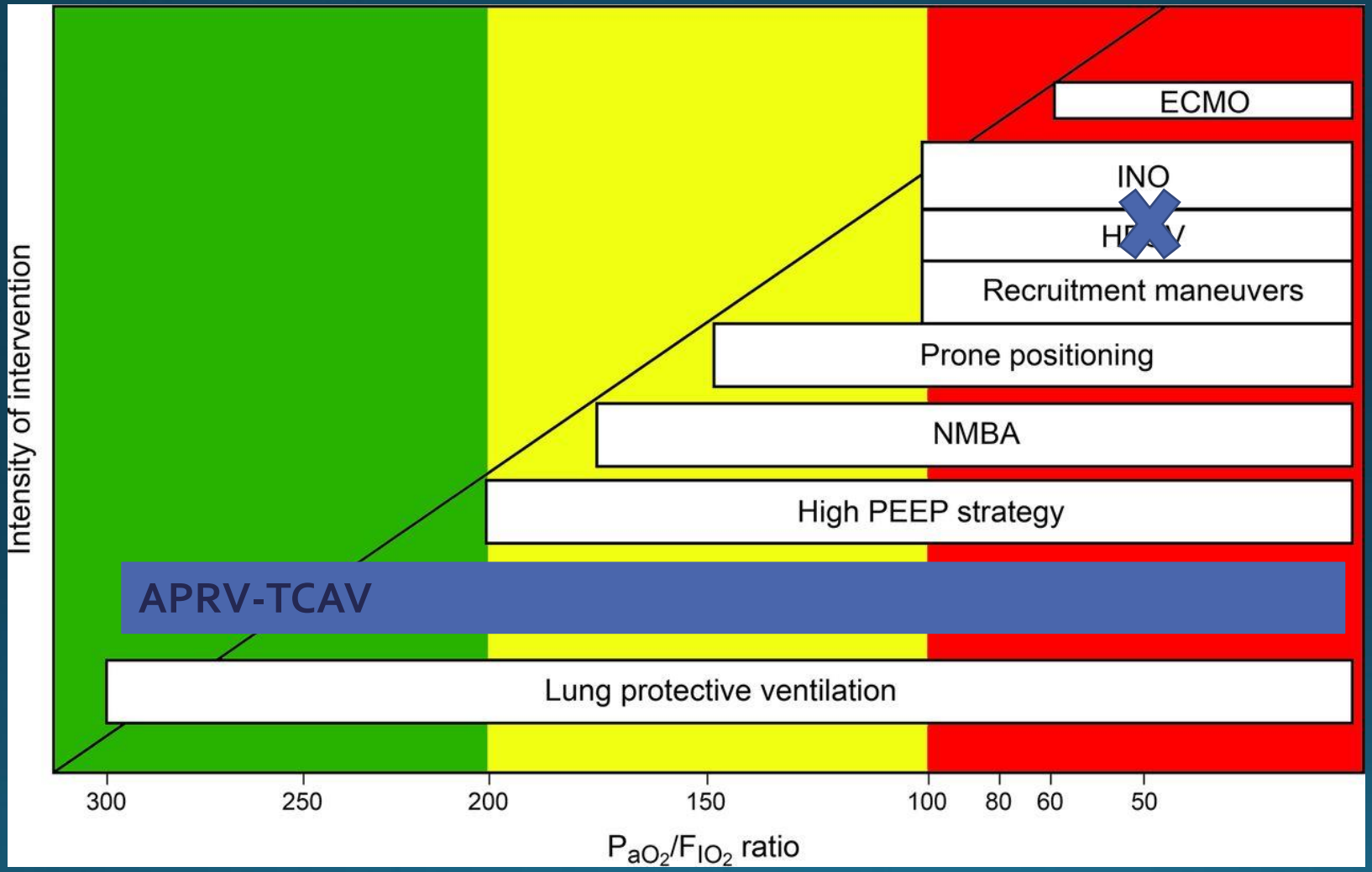
Crit Care Med 2019 Epub

Airway Pressure Release Ventilation in Adult Patients With Acute Hypoxemic Respiratory Failure: A Systematic Review and Meta-Analysis

Jolene Lim, MBBS (Hon), MSc (Dist)[1]; Edward Litton, MBChB, FCICM, MSc, PhD[1,2]

Conclusions: In adult patients requiring mechanical ventilation for acute hypoxic respiratory failure, airway pressure release ventilation is associated with a mortality benefit and improved oxygenation when compared with conventional ventilation strategies. Given the limited number of patients enrolled in the available studies, larger multicenter studies are required to validate these findings. (*Crit Care Med* 2019; XX:00–00)

Rescue therapy in increasing hypoxemia severity.



Francesco Alessandri et al. Respir Care 2018;63:92-101

RESEARCH ARTICLE | OCTOBER 21 2020

A Pilot Study of Patients With COVID–19-Related Respiratory Failure Utilizing Airway Pressure Release Ventilation (APRV)

D'Andrea K. Joseph ; Gerard A. Baltazar ; Ricardo A. Jacquez ; Shahidul Islam ; Adam Stright ; Jasmin Divers ; Collin E.M. Brathwaite ; Patrizio Petrone

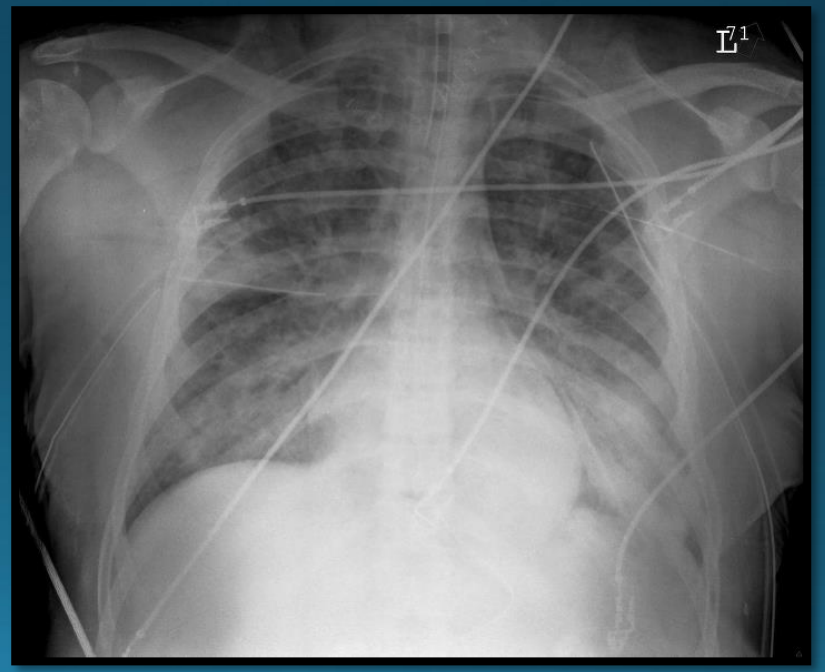
Innovations in Surgery and Interventional Medicine (2021) 1 (1): 3–8.

https://doi.org/10.36401/ISIM-20-03 Article history

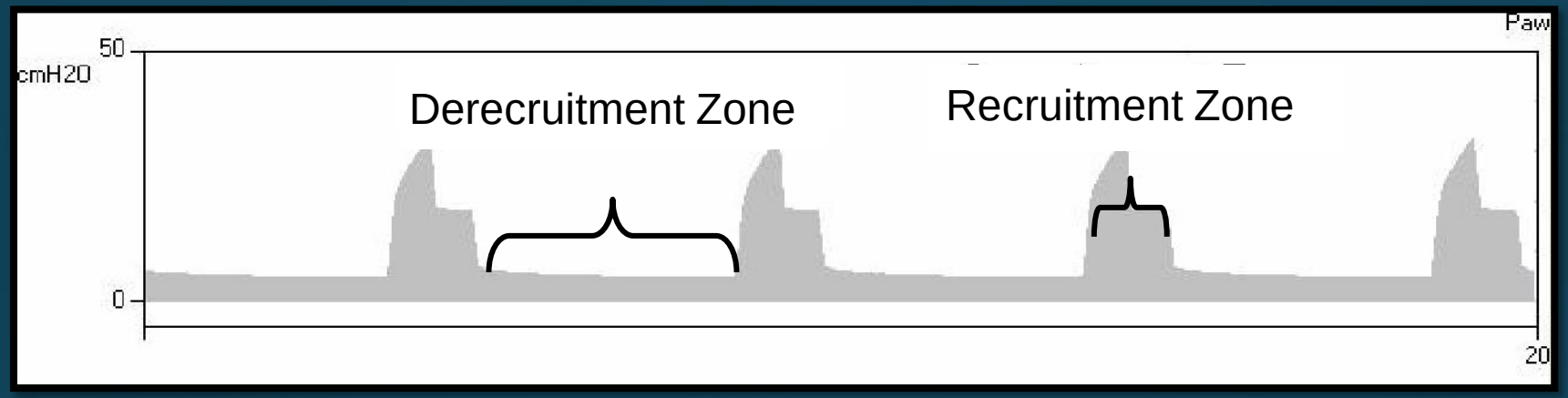
Indications for APRV

- Intubation with the goal of minimizing sedation and preservation of spontaneous breathing

- Differing disease states may require modified settings
  - Recruitable versus
  - Non-Recruitable



Recruitment vs Derecruitment Zones

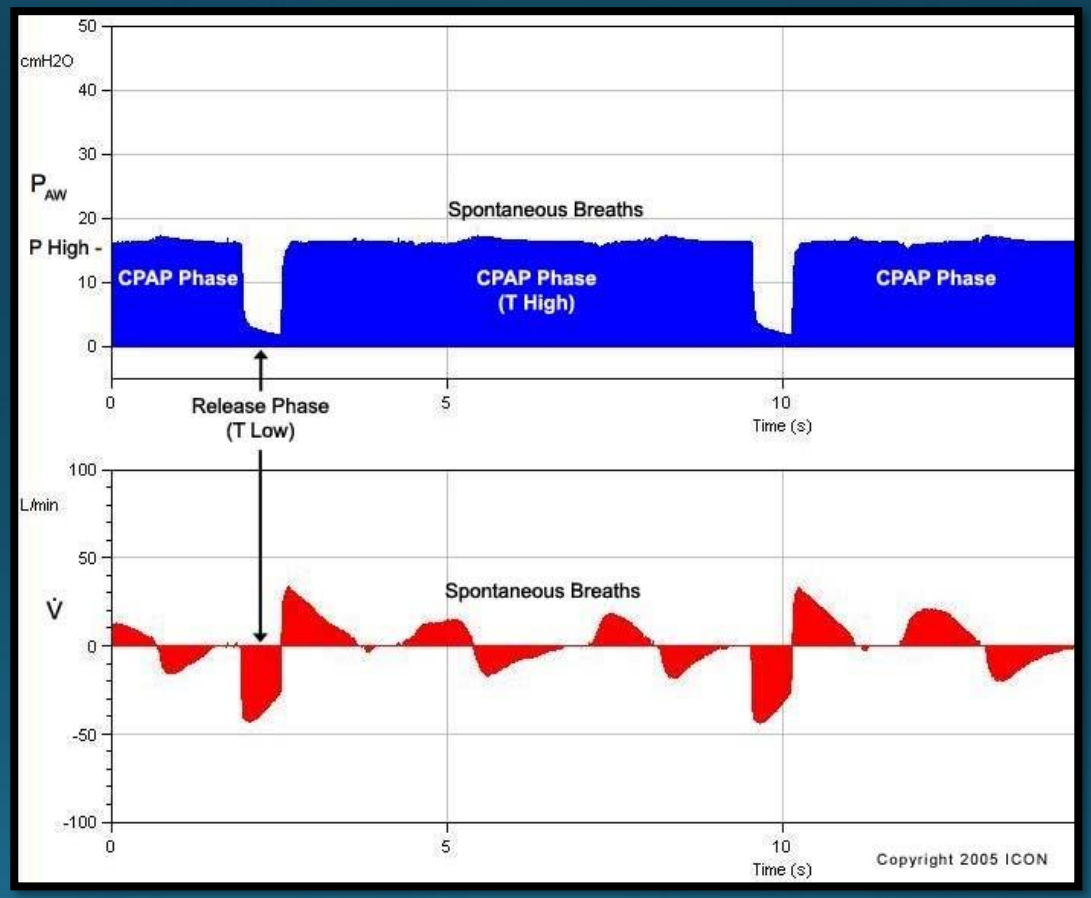


Contraindications for APRV

- Unfamiliarity
- Knowledge Deficit

APRV SETTINGS

- P High
- P Low – PEEP
- T High
- T Low
- FiO_2
- Slope/Rise Time
- ATC/PS



SLOPE/RISE TIME

- Slope typically set to 0 seconds to maximize inspiratory time
- May need to increase with smaller artificial airway

P High

P High

- P High Goal
  - enough pressure to open lung but not over-distend
  - Between FRC and TLC

P High (Adult)

- Using as Initial Mode Upon Intubation
  - Typically 21-24 cmH_2O
  - Adjust as necessary based on oxygenation and ventilation
  - Assess your settings

- Transitioning from a [Pure] Volume Mode
  - Set at current plateau pressure
  - (not mean airway pressure)

- Transitioning from a Pressure or Dual Targeted Mode
  - Set at current total pressure from pressure mode

Assessing intra-vascular Status
Pre-load Dependency Test

- Mean Airway pressure in APRV usually ~3 cm H2O below P High
- Adequate vascular volume is important to maintain adequate perfusion through the lungs

- How to asses vascular status
  - Passive Leg Raise
  - Liver compression
  - Trendelenburg
  - Increase PEEP

- APRV creates a high mean airway pressure

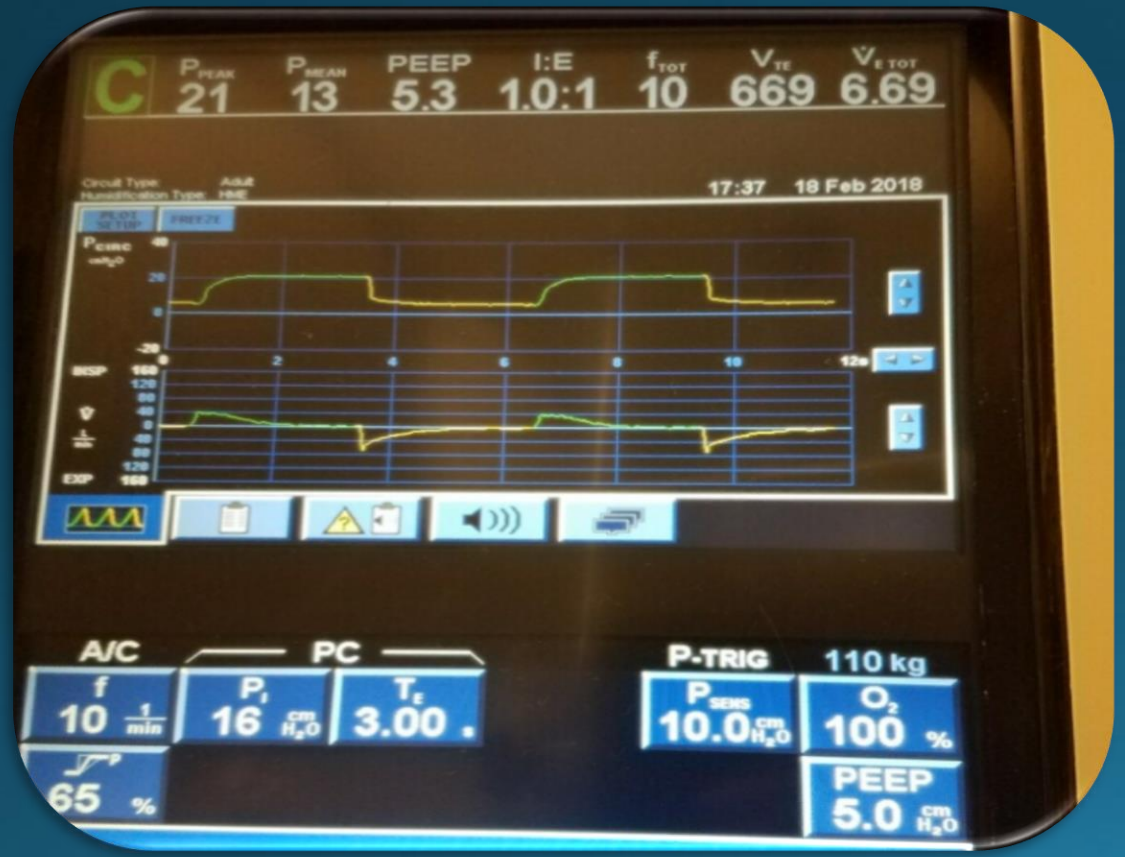
Nieman *et al. Critical Care* (2018) 22:136
https://doi.org/10.1186/s13054-018-2051-8

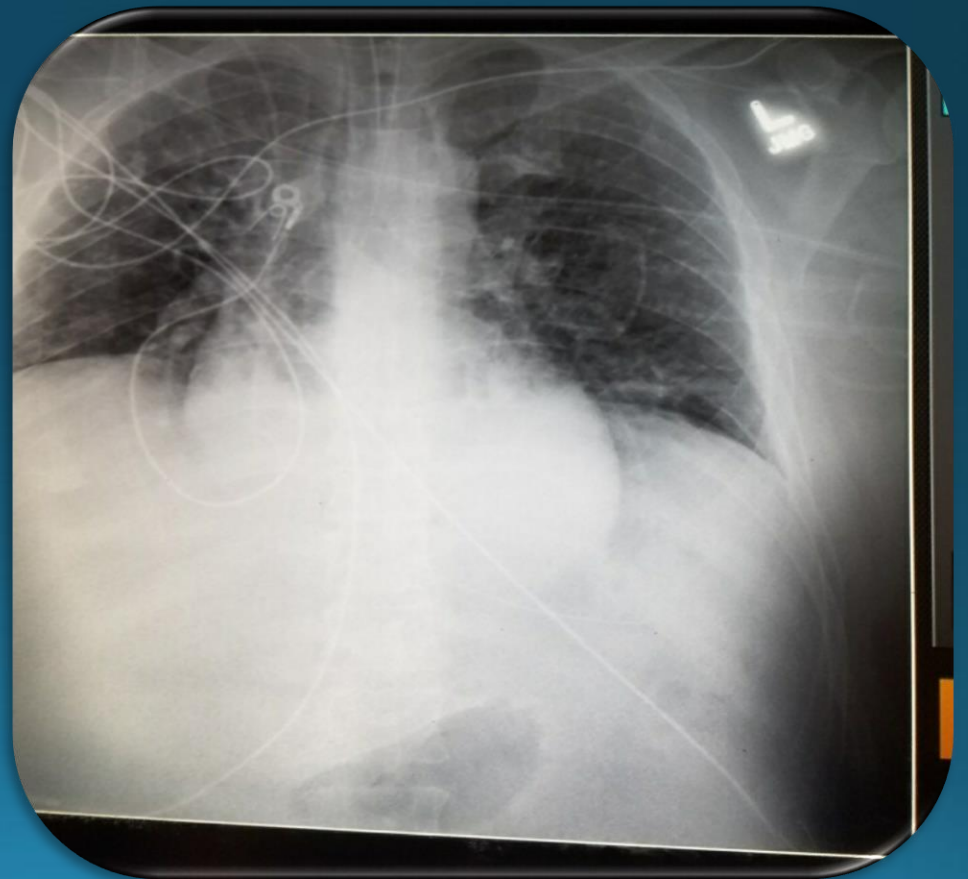
Critical Care

VIEWPOINT Open Access

Acute lung injury: how to stabilize a broken lung

Gary F. Nieman[1], Penny Andrews[2], Joshua Satalin[1*], Kailyn Wilcox[1], Michaela Kollisch-Singule[1], Maria Madden[2], Hani Aiash[1], Sarah J. Blair[1], Louis A. Gatto[1,3] and Nader M. Habashi[2]

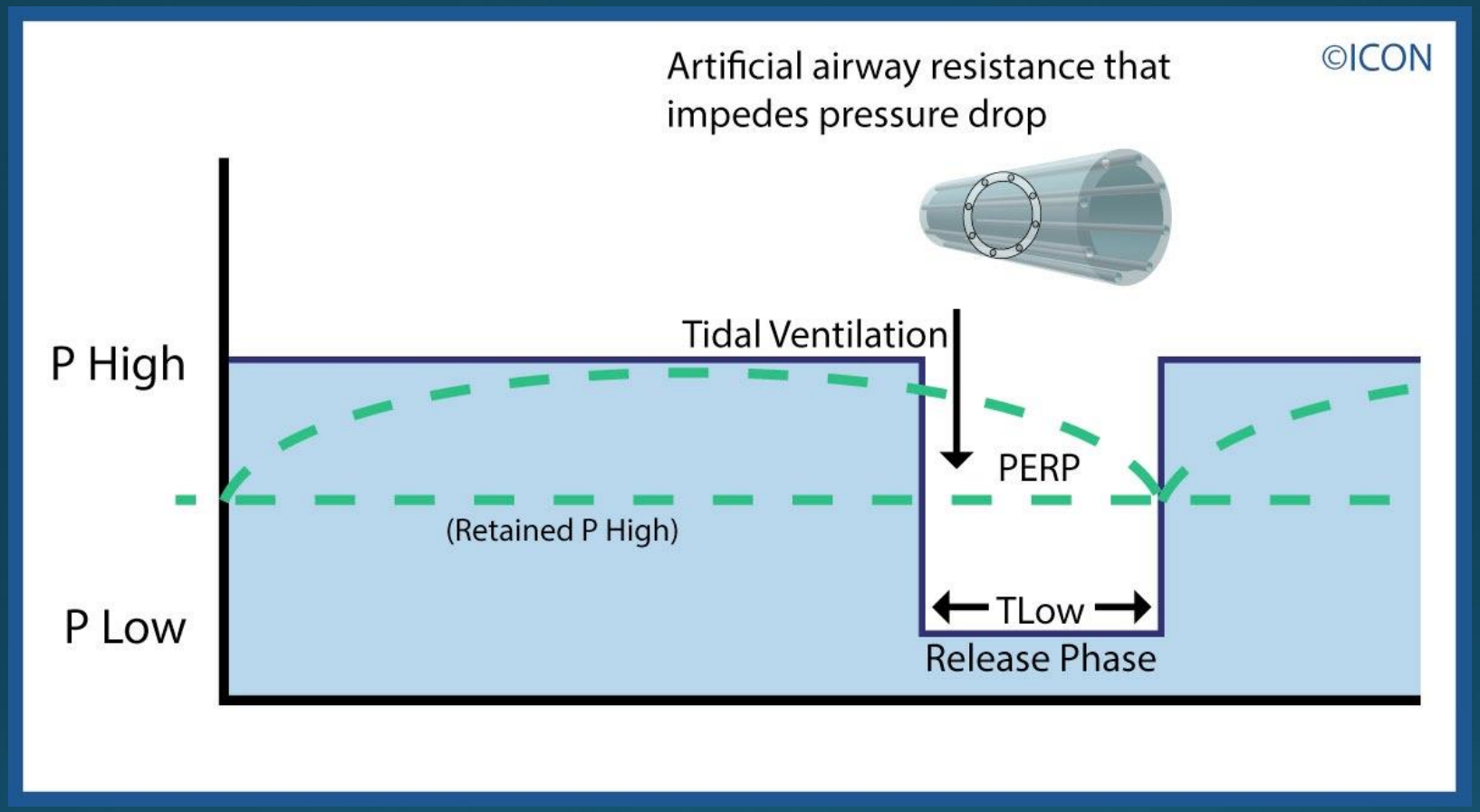


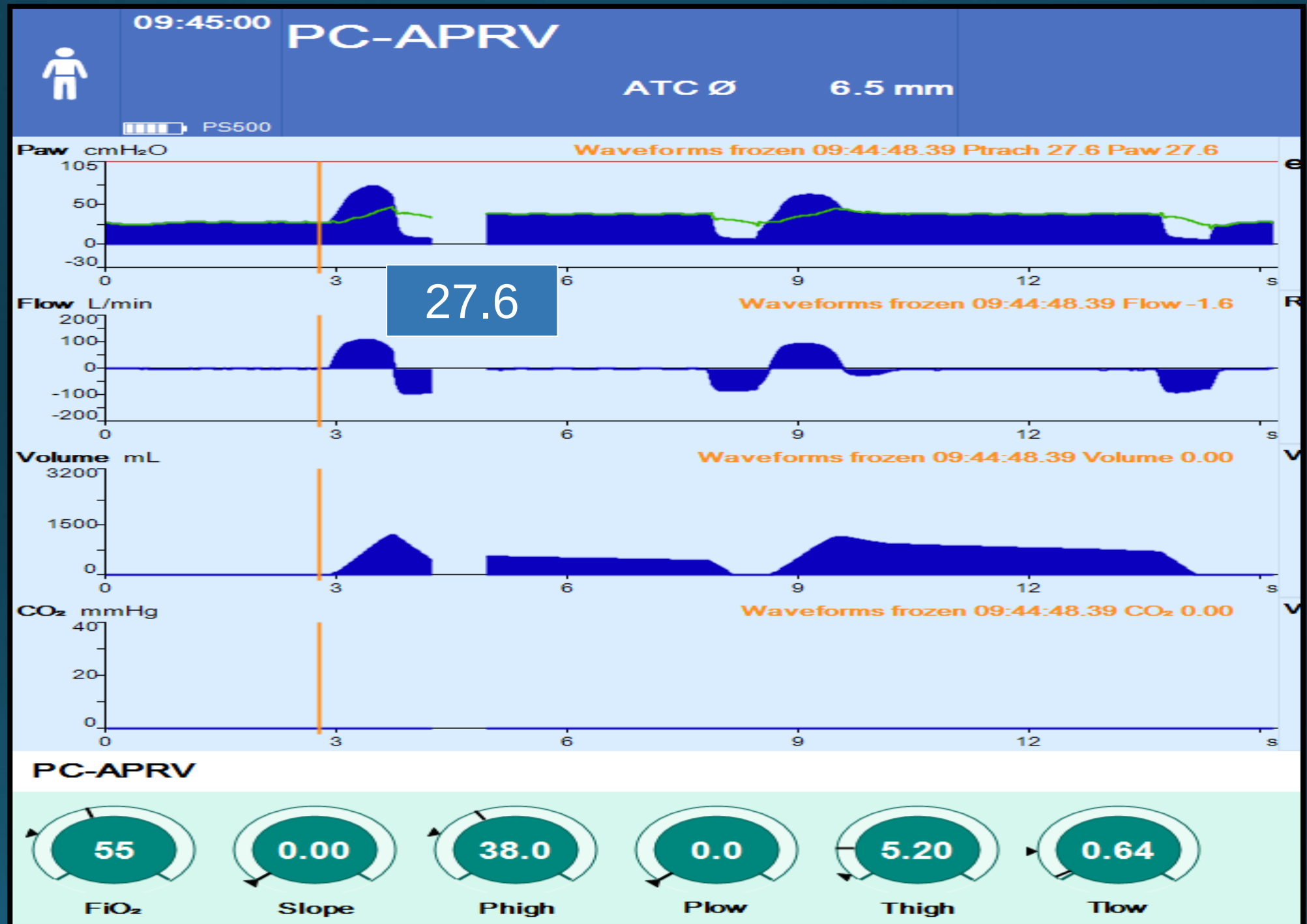


P LOW

P Low

- P Low set at o cmH_2O
- When using a P Low of o **MUST** have T Low set appropriately
- Decreases expiratory resistance
- Controlling end expiratory lung volume with time rather than pressure
- P Low also known as PEEP on some ventilators
- We are creating PEEP based on the patient's chart not


Artificial airway resistance that impedes pressure drop
©ICON
Tidal Ventilation
P High
PERP
(Retained P High)
P Low
TLow
Release Phase


09:45:00
PC-APRV
ATC Ø 6.5 mm
PS500
Paw cmH₂O
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CO₂ mmHg
Waveforms frozen 09:44:48.39 CO₂ 0.00
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PC-APRV
55
FiO₂
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38.0
Phigh
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Plow
5.20
Thigh
0.64
Tlow

SPECIAL ARTICLE

Driving Pressure and Survival in the Acute Respiratory Distress Syndrome

Marcelo B.P. Amato, M.D., Maureen O. Meade, M.D., Arthur S. Slutsky, M.D., Laurent Brochard, M.D., Eduardo L.V. Costa, M.D., David A. Schoenfeld, Ph.D., Thomas E. Stewart, M.D., Matthias Briel, M.D., Daniel Talmor, M.D., M.P.H., Alain Mercat, M.D., Jean-Christophe M. Richard, M.D., Carlos R.R. Carvalho, M.D., et al.

Comparing Driving Pressures in Airway Pressure Release Ventilation in Trauma Intensive Care Unit Patients

Penny Andrews[1], Maria Madden[1], Josh Satalin[2], Gary Nieman[2], Nader Habashi[1]

After collecting ΔP measurements on 200 trauma subjects, the data was divided among the four categories of ventilator modes:

1) Volume Control (VC)

2) Pressure Control (PC)

3) Airway Pressure Release Ventilation (APRV) using the Time Controlled Adaptive Ventilation (TCAV) Method

Comparing Driving Pressures in Airway Pressure Release Ventilation in Trauma Intensive Care Unit Patients

Penny Andrews[1], Maria Madden[1], Josh Satalin[2], Gary Nieman[2], Nader Habashi[1]

R Adams Cowley Shock Trauma Center
University of Maryland

BACKGROUND

Recent data in a meta-analysis of randomized controlled trials of acute respiratory distress syndrome (ARDS) suggest that driving pressure (ΔP) may be a critical factor in outcome, with a ΔP less than 14 cmH_2O being a better predictor of survival. This is contrary to the original belief that tidal volume (Vt) and plateau pressure (Pplat) were the key determinants of survival benefit.

Previous studies have focused on ΔP; however, little to no data exist for ΔP with the mode Airway Pressure Release Ventilation (APRV) in trauma intensive care unit (ICU) patients. Our aim with this study was to assess ΔP among our trauma ICU patients in a variety of modes of mechanical ventilation including APRV.

METHODS & MATERIALS

This was an observational, prospective study approved by the University of Maryland Medical Center (UMMC) R Adams Cowley Shock Trauma Center (STC) Institutional Review Boards (IRBs). Patients were screened and data was collected on 200 patients receiving mechanical ventilation at the UMMC-STC in the intensive care units (ICUs) including the multi-trauma ICU, neuro-trauma ICU , critical care resuscitation unit (CCRU) and the lung recovery unit (LRU). Eligibility criteria were: 1) patients 18 years or older, 2) receiving mechanical ventilation with an artificial airway, and 3) not spontaneously breathing [for reasons that included clinical and/or pharmacological intervention with neuromuscular blocking agents, deep sedation or post-operatively].

METHODS & MATERIALS

reversal. During ΔP measurements, patients were assessed for spontaneous breathing efforts using waveform graphics and physical assessment to ensure there were no efforts made.

The ΔP was calculated by subtracting PEEP from the plateau pressure (Pplat). In the conventional ventilation group, the Pplat was measured after a 2-3 second inspiratory hold and the PEEP level was measured with a 4 second expiratory hold to assure there was no auto-PEEP (Figure 1). In the APRV group, the Pplat was validated with a 4 second inspiratory hold. An expiratory hold was used for 4 seconds to allow pressure equilibrium during the release phase to measure PEEP (Figure 2).

DATA

After collecting ΔP measurements on 200 trauma subjects, the data was divided among the four categories of ventilator modes: 1) Volume Control (VC) n=86; 2) Pressure Control (PC) n=28; 3) Airway Pressure Release Ventilation (APRV) using the Time Controlled Adaptive Ventilation (TCAV) Method as standard of care (S-APRV) n=74; and 4) APRV-TCAV as a rescue mode (R-APRV) n=12 used when patients failed other conventional modes of mechanical ventilation and were transitioned to APRV with the TCAV method.

RESULTS

significant difference compared to the VC group (p-value=0.0010) and PC group (p-value=0.0002) but not statistically different than R-APRV group (p-value=0.3379).

CONCLUSION

The ΔP in S-APRV was significantly lower than conventional modes VC and PC.

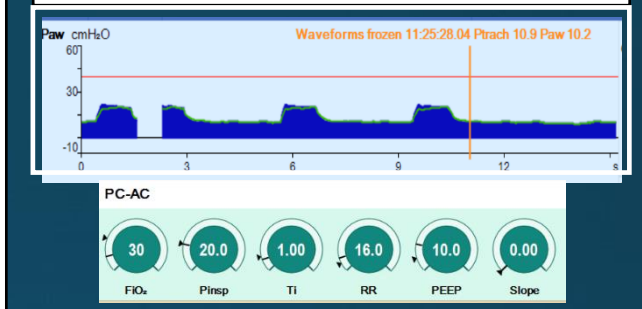


Figure 1 – Expiratory hold – PEEP measurement in PC-AC

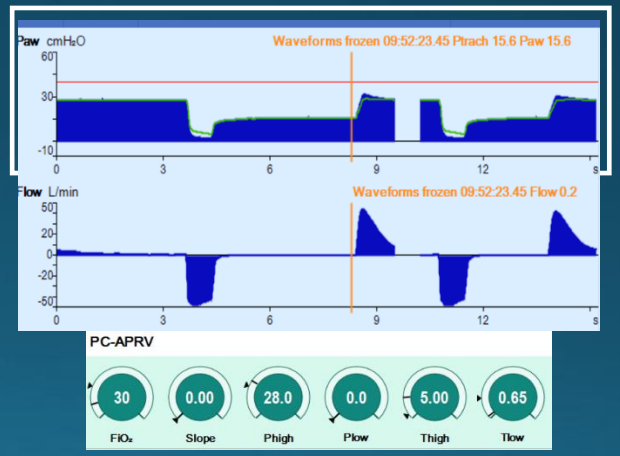


Figure 2 – Expiratory hold – PEEP measurement in APRV

DISCLOSURE

MM, PA, JS, GN, NH have lectured at Dräger sponsored conferences and ICON. MM, PA are employed by ICON and NH has conducted consulting

Comparing Driving Pressures in Airway Pressure Release Ventilation in Trauma Intensive Care Unit Patients

Penny Andrews[1], Maria Madden[1], Josh Satalin[2], Gary Nieman[2], Nader Habashi[1]

	APRV	VC	PC
Driving Pressure	10.65	12.36	13.37
Mortality %	23	25	30

LUNG RECRUITMENT DECREASES DRIVING PRESSURE, NOT THE CLINICIAN

Maria Madden, Penny Andrews , Nader Habashi

BACKGROUND

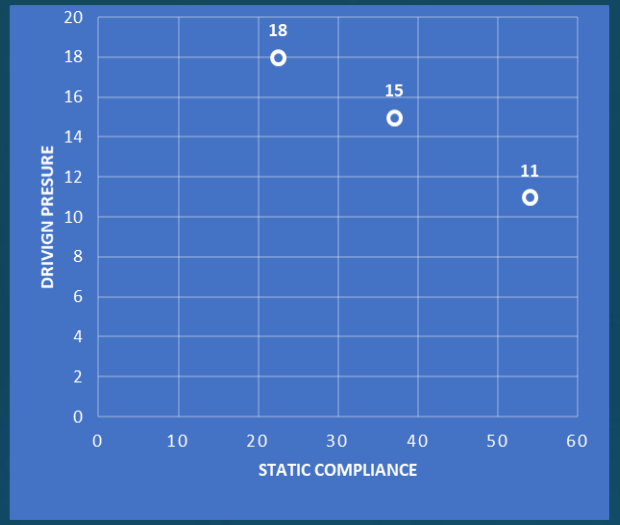
Recent studies demonstrate that a driving pressure (ΔP) <14 cmH_2O improves outcome in acute respiratory distress syndrome (ARDS). Although clinicians may target a desired ΔP by adjusting the ventilator settings (i.e. increasing PEEP, lowering tidal volumes (Vt), etc), DP may be used as a tool to monitor to detect lung recruitment where lung compliance [and Vt] improves without ventilator adjustments. In a 2015 paper, Amato et al. state: "Because respiratory-system compliance (C_{RS}) is strongly related to the volume of aerated remaining functional lung during disease (termed functional lung size), we hypothesized that driving pressure ($\Delta P=V_T/C_{RS}$), in which V_T is intrinsically normalized to functional lung size (instead of predicted lung size in healthy persons), would be an index more strongly associated with survival than Vt or PEEP in patients who are not actively breathing". In conclusion, they state "We found ΔP was the ventilation variable that best stratified risk".

In this case, we present the evolution of ΔP as it decreased and its correlation with recruitment and improvement in C_{RS} in a patient diagnosed with severe ARDS based on Berlin criteria.

Amato M, Meade M, Slutsky A, et al. Driving Pressure and Survival in the Acute Respiratory Distress Syndrome. N Engl J Med 2015;372:747-755.

CASE STUDY

A 35-year-old man with a body mass index (BMI) of 36.7 who initially presented at an outside hospital with hypoxia, fever, and positive blood cultures. He required intubation, vasoactive agents for hemodynamic instability, multiple antibiotics, and inhaled pulmonary vasodilator (Flolan) for severe hypoxemia. The chest radiograph was read as diffuse opacities throughout both lungs with a pneumomediastinum and right pneumothorax. The patient's condition worsened, and he was transferred to the University of Maryland Medical Center - R Adams Cowley Shock Trauma Center (STC) for consideration of extracorporeal membrane oxygenation(ECMO).



Upon admission to STC, he was evaluated for ECMO and based on religious beliefs was deemed inappropriate. After transfer, he was transitioned to Airway Pressure Release Ventilation (APRV) using the Time Controlled Adaptive Ventilation (TCAV) method. The initial ΔP was 18 cmH_2O on TCAV-APRV. Subsequently, all vasoactive medications and Flolan were weaned off despite an increase in mean airway pressure.and over the next 24 hours decreased to 15 cmH_2O and was 11 cmH_2O after 48 hours. On Day 9 of his admission to STC, he was transitioned to CPAP and extubated the following day.

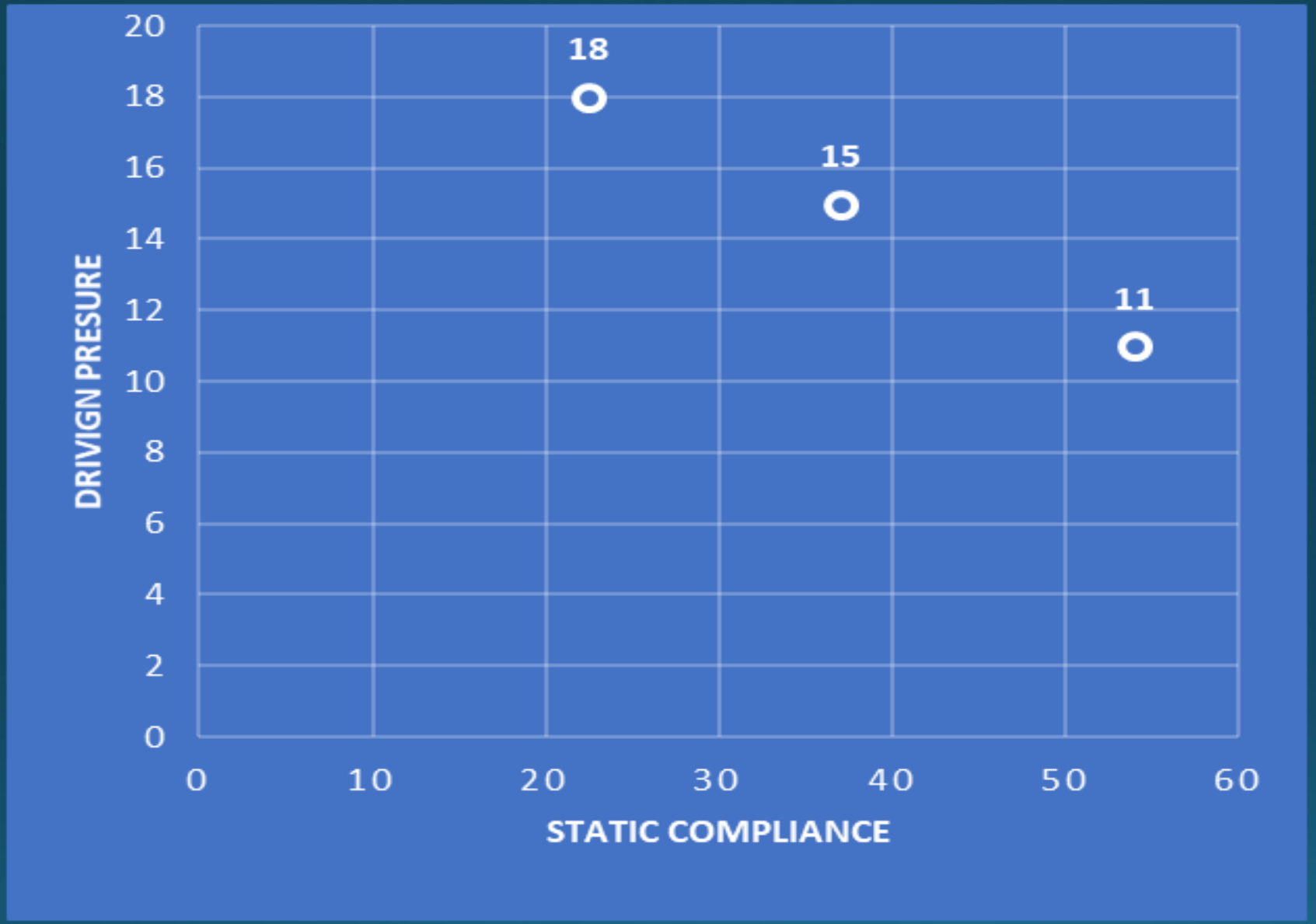
DISCUSSION

Although the initial driving pressure was 18 cm H_2O it decreased over time despite unchanged ventilator settings on APRV and correlated with an improvement in compliance (see Figure 1), clearing chest radiographs, improvement in P/F ratio. This case demonstrates the use of DP as a tool to trend an improvement in recruitment (compliance) with optimal ventilator settings versus changing ventilator settings to decrease driving pressure.

DATE	DRIVING PRESSURE cm H2O	P/F RATIO	VENTILATOR SETTINGS	MEDICATIONS
12/30/17 1420 OSH		46	Volume Control RR 22 BPM VT 420 ml PEPP 16 1.0 FIO2 Transitioned to APRV	Norepinephrine Epinephrine Inhaled Flolan
1/4/18	18	103	33/0 4.5/0.5 55%	All the above medications discontinued
1/5/18	15	223	31/0 4.7/0.59 50%	
1/6/18	11	338	29/0 4/0.6 40%	

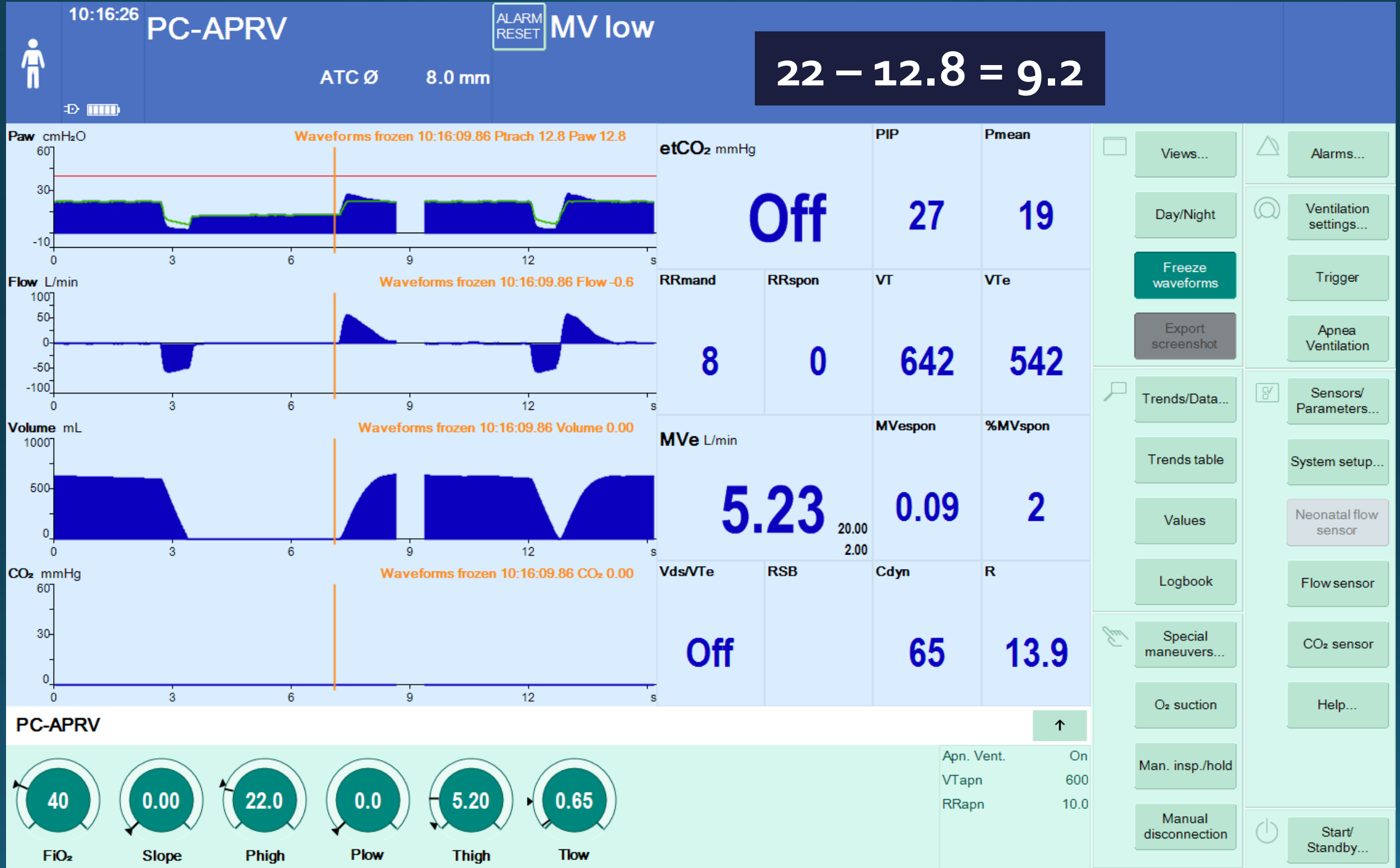
Disclosure

MM, PA,, NH have lectured at Dräger sponsored conferences and ICON. MM, PA are employed by ICON and NH has conducted consulting work with ICON. NH holds several patents related to mechanical ventilation



Extubated 11 days from admission

Why aren't we using APRV-TCAV before V V ECMO for ARDS?


10:16:26
PC-APRV
ALARM RESET
MV low
ATC Ø
8.0 mm
22 – 12.8 = 9.2
Paw cmH₂O
Waveforms frozen 10:16:09.86 Ptrach 12.8 Paw 12.8
Flow L/min
Waveforms frozen 10:16:09.86 Flow -0.6
Volume mL
Waveforms frozen 10:16:09.86 Volume 0.00
CO₂ mmHg
Waveforms frozen 10:16:09.86 CO₂ 0.00
etCO₂ mmHg
Off
PIP
27
Pmean
19
RRmand
8
RRspon
0
VT
642
VTe
542
MVe L/min
5.23
20.00
2.00
MVespon
0.09
%MVspon
2
Vds/VTe
Off
RSB
Cdyn
65
R
13.9
Views...
Day/Night
Freeze waveforms
Export screenshot
Trends/Data...
Trends table
Values
Logbook
Special maneuvers...
O₂ suction
Man. insp./hold
Manual disconnection
Alarms...
Ventilation settings...
Trigger
Apnea Ventilation
Sensors/ Parameters...
System setup...
Neonatal flow sensor
Flow sensor
CO₂ sensor
Help...
Start/ Standby...
PC-APRV
40
FiO₂
0.00
Slope
22.0
Phigh
0.0
Plow
5.20
Thigh
0.65
Tlow
Apn. Vent. On
VTapn 600
RRapn 10.0

Driving Pressure

P High – TC PEEP = Driving Pressure

22 – 12.8 = 9.2

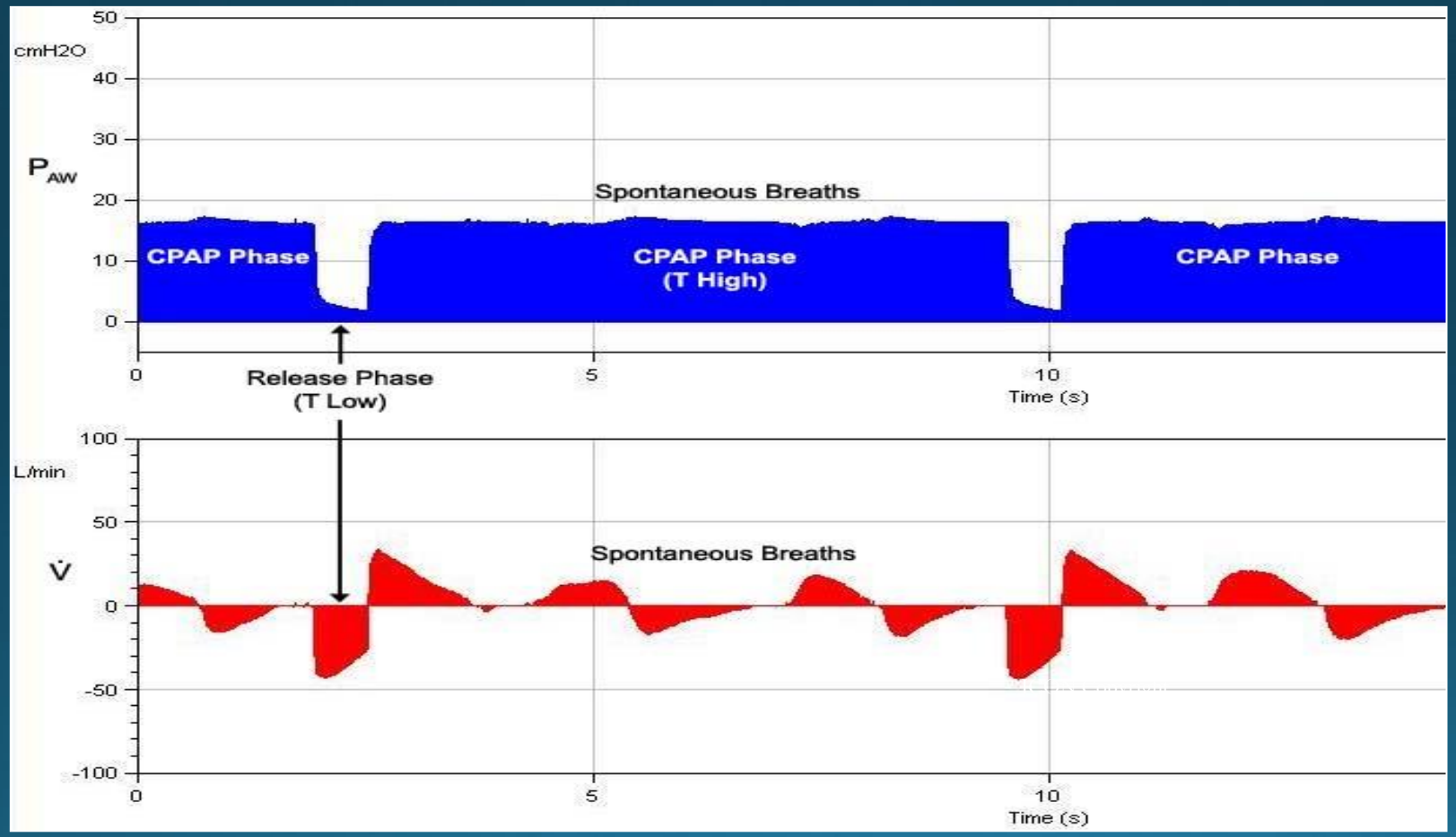
Very important to have T Low set correctly


10:14:43
PC-APRV
ALARM RESET
MV low
ATC Ø
8.0 mm
22 – 15.2 = 6.8
Paw cmH₂O
Waveforms frozen 10:14:17.26 Ptrach 15.2 Paw 15.2
Flow L/min
Waveforms frozen 10:14:17.26 Flow -0.6
Volume mL
Waveforms frozen 10:14:17.26 Volume 0.00
CO₂ mmHg
Waveforms frozen 10:14:17.26 CO₂ 0.00
etCO₂ mmHg
Off
PIP
25
Pmean
20
RRmand
10
RRspon

VT
433
VTe
326
MVe L/min
3.43
20.00
2.00
MVespon
0.00
%MVspon
0
Vds/VTe
Off
RSB
Cdyn
32.7
R
13.3
Views...
Day/Night
Freeze waveforms
Export screenshot
Alarms...
Ventilation settings...
Trigger
Apnea Ventilation
Trends/Data...
Trends table
Values
Logbook
Sensors/ Parameters...
System setup...
Neonatal flow sensor
Flow sensor
CO₂ sensor
Special maneuvers...
O₂ suction
Man. insp./hold
Manual disconnection
Help...
Start/ Standby...
PC-APRV
40
FiO₂
0.00
Slope
22.0
Phigh
0.0
Plow
5.20
Thigh
0.40
Tlow
Apn. Vent. On
VTapn 600
RRapn 10.0

T HIGH

T High – *Duration of CPAP Phase*



T High

- 4 - 6 seconds for Adults
  - 5.0-5.5 is typical
- 90% cycle time is at T High for adults
  - T high / Total Time x 100 =
  - 5/(5 + 0.55) x 100 = 90%
- 80-85% cycle time is at T High for pediatric and neonates
- Recruitment takes ***time***

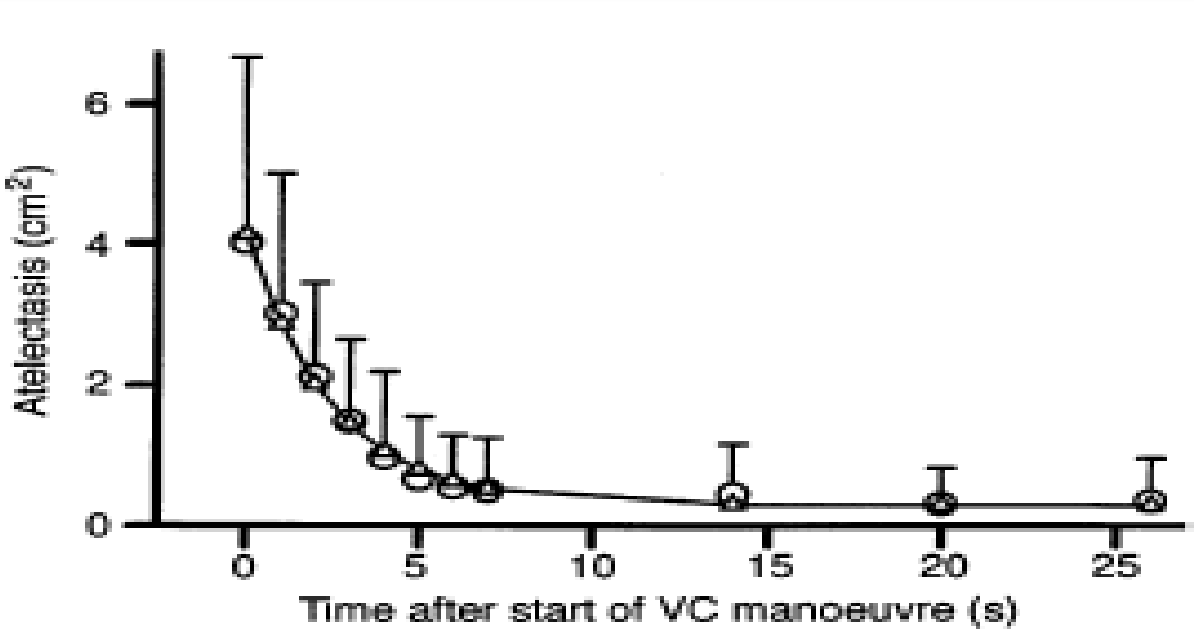


Fig 1 Atelectasis before and during the VC manoeuvre. Mean values (○) and SD (error bars) are shown. Also shown is a curve with negative exponential decay, fitted to individual data (△), connected by a line. For further details, see text.

British Journal of Anesthesia 82 (4): 551-6 (1999)

Calculating Respiratory Rate

- T High is time during CPAP
- T Low is time during release
- 60 seconds/Total cycle time (T High + T Low) = BPM

  - 60/(4+0.7) = 13 BPM
  - 60/(5 + 0.7) = 11 BPM
  - 60/(5 + 0.8) = 10 BPM
  - 60/(6 + 0.7) = 9 BPM

USE OF APRV VENTILATION WITH A TRAUMATIC BRAIN INJURED PATIENT

RC JOURNAL ABSTRACT 2011

	SIMV	APRV
ABG	7.45/35/150/26/98% P/F 272	7.49/32/178/24/1.4/98% P/F 395
RR	25 BPM Tidal Volume 550 ml PEEP 14 FIO2 55%	13 BPM (releases) P High26/ P Low 0 T High 4.2/ T Low 0.55 FIO2 45%
ICP	7-11	4-12

	ADMISSION	DAY 1	DAY 3	DAY 5
MODE	VC-SIMV	APRV	APRV	APRV
FIO2	50%	60%	40%	35%
SETTINGS	RR 22 VT 480 PS 10/PEEP 15	P High 32cm H20 P Low 0 T High 5.5 T Low 0.6 (Set RR @ 10)	P High 28cm H20 P Low 0 T High 6.0 T Low 0.6	P High 24cm H20 P Low 0 T High 6.0 T Low 0.6
PEAK/PLATEAU	40/32	32	28	24
SPONT RR	4	17	0	0
TOTAL RR	26	27	9	9
PACO2	27	37	40	37
P/F RATIO	128	162	413	483

THE USE OF AIRWAY PRESSURE RELEASE VENTILATION (APRV) PREVENTS THE NEED FOR EXTRACORPOREAL MEMBRANE OXYGENATION (ECMO) IN A TRAUMA PATIENT

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University of Maryland Medical Center

R Adams Cowley Shock Trauma Center, University of Maryland

Abstract

INTRODUCTION

Because of the unique prolonged pressure-time profile, Airway Pressure Release Ventilation (APRV) has been described as Continuous Positive Airway Pressure (CPAP) with a brief release phase which augments carbon dioxide (CO_2) removal. Although APRV may be applied as soon as intubation, it is often used as a rescue mode after respiratory failure has progressed acute respiratory distress syndrome (ARDS). Current clinical practice is to apply lung protective strategies such as APRV, low tidal volume ventilation (ARDSnet strategy), recruitment maneuvers, HFOV and extracorporeal membrane oxygenation (ECMO) after respiratory failure progresses to ARDS. However, recent clinical and laboratory data demonstrate that early application of APRV can prevent ARDS. This case study illustrates that early application of APRV not only resolved profound hypoxemia and ARDS but also spared the patient ECMO therapy. Early intervention by the respiratory therapist(s) to place this patient on APRV improved oxygenation and ventilation, restored alveolar stability and prevented ECMO.

DISCUSSION

A 23 year old male was admitted to the R Adams Cowley Shock Trauma Center (STC) after a high speed motor vehicle collision suffering a right tension pneumothorax, bilateral pulmonary contusions, right lung laceration, several orthopedic injuries and a small frontal lobe hemorrhage. He was hypoxemic with P/F ratio of 72 on 100% FiO_2 and in shock as evidenced by hypotension and a lactate level of 7.0 mmol/L. Upon admission to STC, he was initially placed on AC-VC/Autoflow with FiO2 100%, set rate of 22, tidal volumes 6cc/kg Ideal Body Weight (470 mL), PEEP 14 cmH_2O with peak airway pressure of 50 cmH_2O. Because he had suffered two pulseless electrical activity (PEA) events related to refractory hypoxia with a profound respiratory acidosis, it was decided by the primary physician to initiate venous-venous (VV) ECMO. During the circuit set-up for VV-ECMO, the primary respiratory therapist (RT) transitioned the patient to APRV using the plateau pressure on AC/VC. After transition to APRV [three hours after admission], oxygenation and ventilation dramatically improved. Subsequent adjustments to the APRV settings allowed for further reduction of FiO_2 to 60% within 4 hours. The patient received required surgical interventions including a tracheostomy as a result of his injuries and was weaned from APRV to humidified tracheostomy collar. This dramatic improvement in oxygenation and ventilation allowed for improvement in hemodynamic stability and the ability for necessary surgical interventions to be performed.

CONCLUSION

In this case study, APRV was used early in the course of respiratory failure preventing the invasive therapy of VV-ECMO. Early application of APRV allowed for improved alveolar stability facilitating lung recruitment, improving oxygenation and ventilation, thereby improving hemodynamic stability.

Data Table

TIME	MODE	SETTINGS	pH	$PaCO_2$	PaO_2	SPO_2	HCO_3	BE	P/F RATIO
0430	PRVC	FiO_2 100% RR 22 VT470 mL PEEP 14 PIP 50 cmH_2O	7.09	61	72	88%	17	-13.3	72
0730	APRV	FiO_2 97% P High 36 cmH_2O P Low 0 cmH_2O T High 2.0 sec T Low 0.65 sec	7.36	29	216	100%	16	-7.8	223
1600	APRV	FiO_2 47% P High 35 cmH_2O P Low 0 cmH_2O T High 5.0 sec T Low 0.75 sec	7.42	25	141	98%	16	-6.0	300

Introduction

A trauma patient with hypoxemia and in shock was being prepped for ECMO cannulation after failing ARDSNet ventilation strategy. Because of a respiratory acidosis (pH of 7.09 and $PaCO_2$ of 61 mm Hg) and the belief that APRV could not effectively manage CO_2, APRV was not initially considered. During the VV-ECMO circuit set-up, the RT successfully transitioned the patient to APRV with a notable increase in oxygenation, thereby avoiding ECMO therapy.

Case Summary

Despite shock, hypoxemia and respiratory acidosis, this patient was transitioned to APRV for stabilization prior to implementing ECMO therapy. There was an immediate improvement in both oxygenation and ventilation with subsequent radiographic improvement via chest x-ray. Swift reduction in FiO_2 and a substantial reduction in peak airway pressures lead to an improvement in alveolar stability and eliminating the need for ECMO. This patient was weaned off mechanical ventilation within

Conclusion

This case demonstrates that APRV is efficient in CO_2 removal with both diffusive and convective CO_2 clearance and can be used for respiratory failure in a patient with a low pH and high $PaCO_2$. Many clinicians do not use APRV with a pre-existing respiratory acidosis believing that APRV cannot efficiently eliminate CO_2, especially in the absence of spontaneous breathing. Recent APRV data demonstrate an increased alveolar stability with increased alveolar ventilation despite a lower minute volume when set appropriately.

Disclosure

The authors have no conflicts of interest nor research funding, sponsorship, or financial support.

TIME	MODE	SETTINGS	pH	$PaCO_2$	PaO_2	SPO_2	HCO_3	BE	P/F RATIO
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0730	APRV	FiO_2 97% P High 36 cmH_2O P Low 0 cmH_2O **T High 2.0 sec** T Low 0.65 sec	7.36	29	216	100%	16	-7.8	223
1600	APRV	FiO_2 47% P High 35 cmH_2O P Low 0 cmH_2O **T High 5.0 sec** T Low 0.75 sec	7.42	25	141	98%	16	-6.0	300

0430	PRVC	FiO_2 100% RR 22 VT470 mL PEEP 14 PIP 50 cmH_2O	7.09	61	72	88%	17	-13.3	72

60 sec/BPM = T Time
T Time – T Low = T High

60/22 = 2.7 T Time
2.7-0.5 = 2.2 T High
T High 2.2/T Low 0.5 (assess waveform) = 22

T High 2.0 sec/T Low 0.65
60 sec/ 2.65 = 23 BPM

0730	APRV	FiO_2 97% P High 36 cmH_2O P Low 0 cmH_2O T High 2.0 Sec T Low 0.65 sec	7.36	29	216	100%	16	-7.8	223

T LOW

T Low

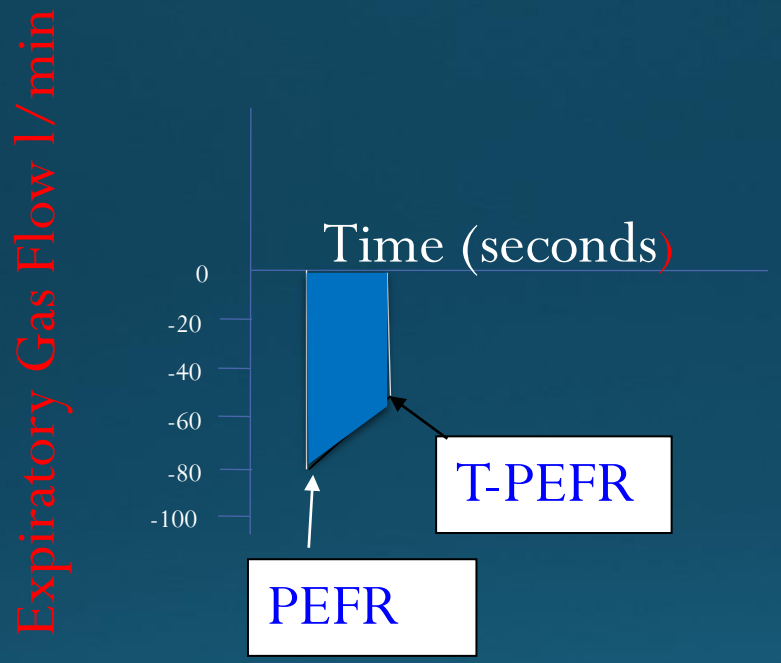
- Controlling "PEEP" with ***time*** rather than ***pressure***
- Varies from patient to patient
  - Based on lung volume and thoracic recoil
- Can also vary based on ventilator type

T Low – Adults/Large Pediatrics

T Low

- 0.5-0.7 seconds ("normal")
- 0.3 - 0.5 seconds (RLD)
- 0.8 - 1.5 seconds (OLD) (25% to 50%)

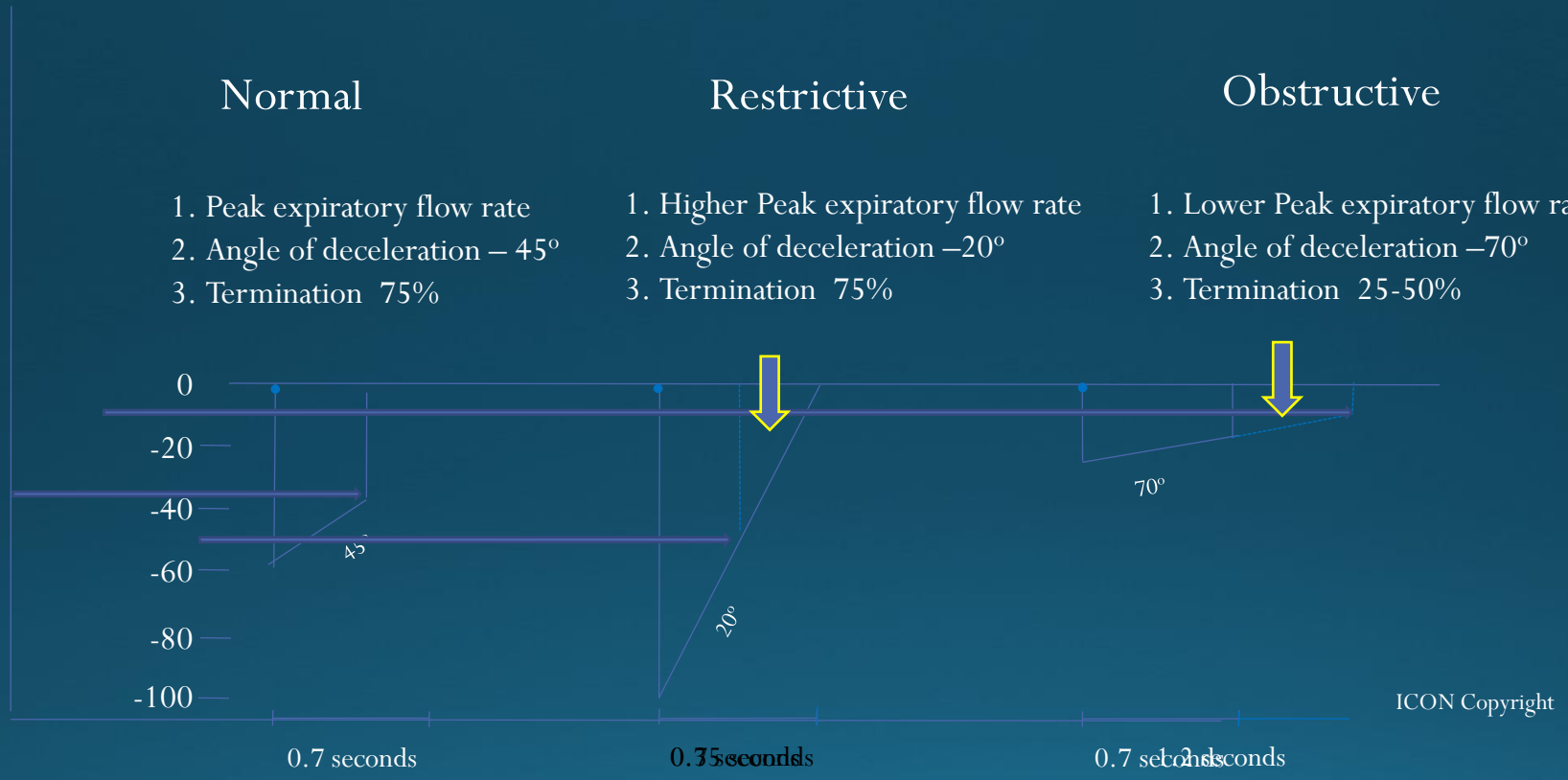
T Low – End Expiratory Lung Volume



T LOW SETTING
T-PEFR/PEFR X 100 = 75%
70 – 75%

60/80 X 100 = 75%

T Low – Retaining End Expiratory Lung Volume



AIRWAY PRESSURE RELEASE VENTILATION MAINTAINS ALVEOLAR STABILITY BY LIMITING LOSS OF LUNG VOLUME DURING RELEASE PHASE

N. Habashi, P. Andrews - R Adams Cowley Shock Trauma Center - University of Maryland School of Medicine, Baltimore, MD;
S. Roy, J. Satalin, K. Snyder, L. Gatto, G. Nieman - SUNY Upstate, Syracuse, NY

INTRODUCTION

Airway Pressure Release Ventilation (APRV) is a mode of mechanical ventilation that has shown promise in prevention and treatment of the acute respiratory distress syndrome (ARDS) when applied early. The P High in APRV equates to a plateau pressure. However, unlike most modes, APRV integrates within the P High tidal gas ventilation and the ability to retain a portion of the P High as positive end release pressure (PERP) similar to PEEP. As the P High is released towards the P Low, expiratory gas flow creates tidal ventilation. Because the duration of the T Low (release phase) is brief (sub-second) and confined to the initial phase of the expiratory release phase, the majority of the resistance impeding the pressure drop from P High towards P Low is created by the artificial airway (Fig. 1). This brief T Low duration prevents alveolar collapse without the need to set PEEP (i.e. P Low of 0 cmH2O) at the expiratory valve. Because of this unique integration, the T Low in APRV uses time to control the release phase and retains a portion of the P High which maintains adequate end-expiratory lung volume, promoting alveolar stability. Therefore, critical to protecting the lung, appropriate APRV application requires optimal adjustment of the T Low. We hypothesized that optimization of the T Low to limit lung volume loss during the release phase would occur at a specific point during Termination of the Expiratory Flow Rate. Specifically, termination at 75% of the Peak Expiratory Flow Rate (T-PEFR/PEFR) would retain the greatest alveolar stability and lower ratios would not achieve alveolar stability leading to greater alveolar volume change and collapse (Fig. 2).

METHODS

In-vivo microscopic fields (n=9) were prepared in anesthetized, male Sprague-Dawley rats. ARDS was induced by instilling 0.2% Tween-20 via tracheostomy. T-PEFR:PEFR was set at 10% and video in-vivo alveolar microscopy performed for multiple respiratory cycles. This procedure was repeated at progressive T-PEFR:PEFR of 25%, 50%, and 75% by decreasing the T Low respectively (Fig. 3).

Quantification of alveolar stability was measured using image analysis software to determine the percent of inflated alveoli occupying the microscopic field at inspiration and at expiration (Fig. 4).

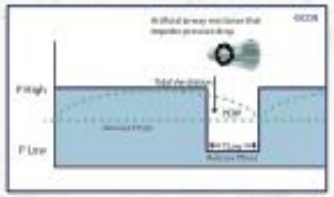


Fig 1. Integration of ventilation and positive end release pressure (PERP) within the P High. The artificial airway creates a majority of the resistance impeding the pressure drop from P High towards P Low. The T Low uses time to retain a portion of the P High, thus retaining adequate end-expiratory lung volume.

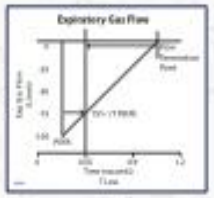


Fig 2. Expiratory gas flow pattern during APRV demonstrating adjustment of the T Low to terminate the expiratory gas flow at 75% of the Peak Expiratory Flow Rate (T-PEFR:PEFR)

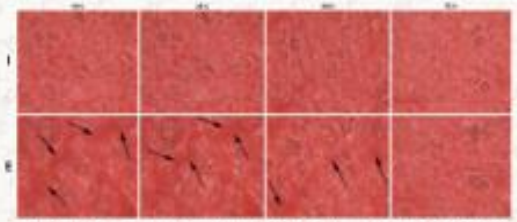


Fig 3. Alveolar Microscopy at 4 different T-PEFR:PEFR percentages. As T-PEFR:PEFR percentage increases, alveolar stability increases.
I=Inspiration E=Expiration
Black arrows illustrate interstitial expansion and alveolar collapse between aerated alveoli at expiration. A progressive decrease in interstitial expansion and greater number of recruited alveoli occupying the field is seen moving left to right from 10% to 75%.

ALVEOLAR STABILITY

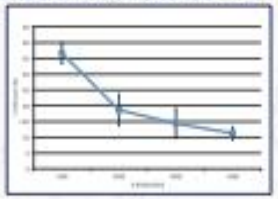


Fig 4. I-E percent alveolar areas were calculated per photomicrographic frame as the difference between the percentage of alveolar area at inspiration and its counterpart at expiration changed. Quantification of alveolar stability shows as T-PEFR:PEFR percentage increases, the alveolar I-E change decreases thus increasing alveolar stability.

RESULTS

T-PEFR:PEFR of 75% had the least alveolar volume change at expiration (10.0%) while T-PEFR:PEFR of 10%, 25% and 50% had a progressively greater alveolar volume change and collapse at expiration: 54.5%, 36.4%, and 29.4% respectively ($p<0.001$ vs T-PEFR 75%) (Fig 5).

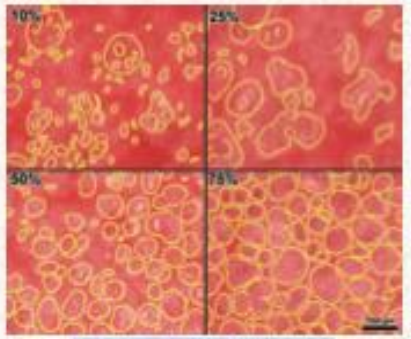
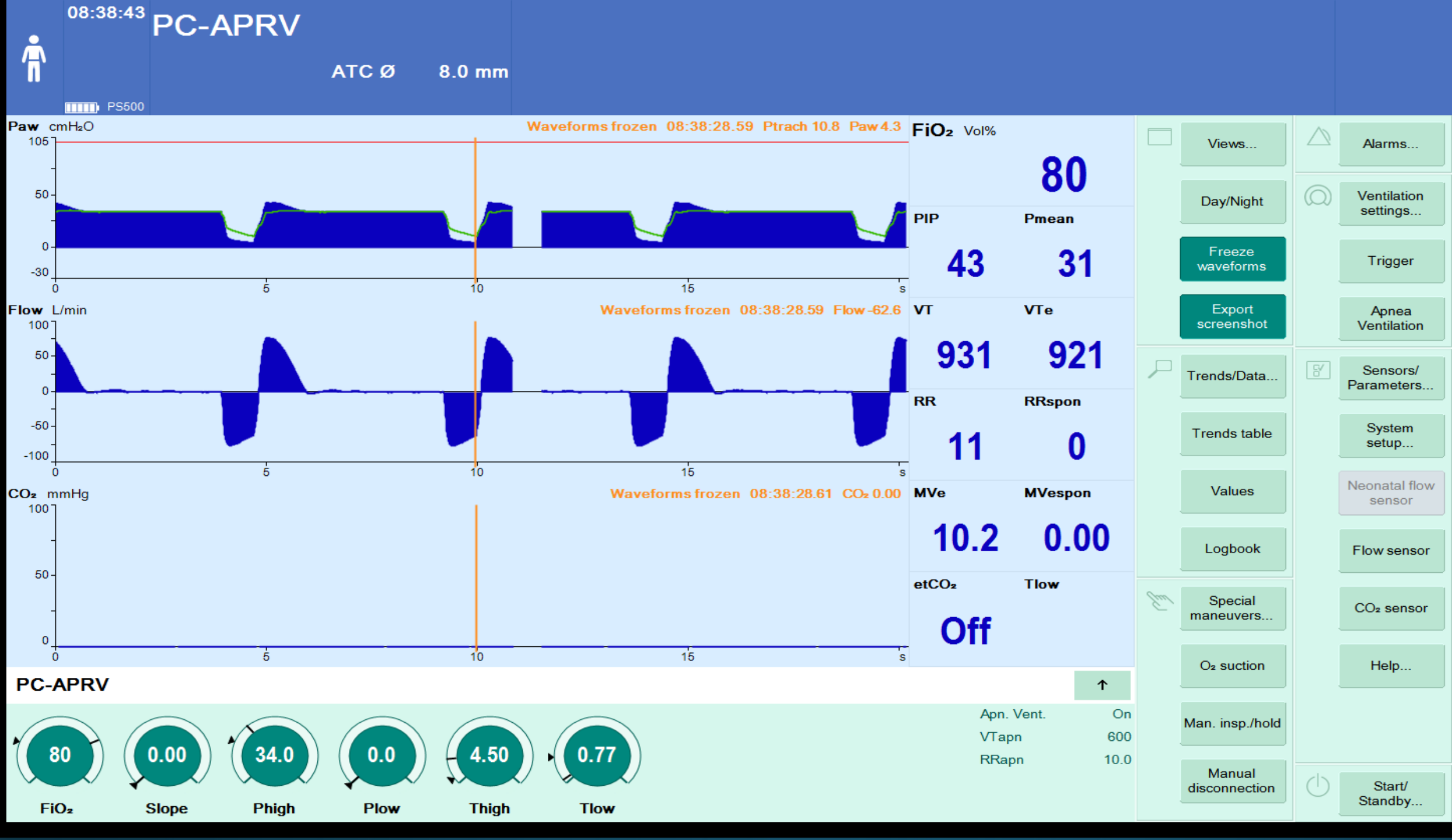


Fig 5. In vivo microscopic fields at expiration with all 4 T Low settings (T-PEFR:PEFR). Alveoli were outlined (yellow) and their collective area was calculated by digital image analysis as a percentage of the total tissue area in the frame.

CONCLUSIONS

The use of APRV as a strategy for the treatment or prevention of ALI/ARDS is contingent on the appropriate method of application. One key aspect of APRV is optimizing the T Low to control expiratory gas flow to minimize alveolar volume change/collapse and maintain alveolar stability. These data confirm that a T-PEFR:PEFR of 75% is necessary to achieve alveolar stability and that a T-PEFR:PEFR <75% may lead to alveolar collapse and lung derecruitment in acute lung injury.


08:38:43
PC-APRV
ATC Ø 8.0 mm
PS500
Paw cmH₂O
Waveforms frozen 08:38:28.59 Ptrach 10.8 Paw 4.3
105
50
0
-30
0
5
10
15
s
Flow L/min
Waveforms frozen 08:38:28.59 Flow -62.6
100
50
0
-50
-100
0
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10
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s
CO₂ mmHg
Waveforms frozen 08:38:28.61 CO₂ 0.00
100
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15
s
FiO₂ Vol%
80
PIP
43
Pmean
31
VT
931
VTe
921
RR
11
RRspon
0
MVe
10.2
MVespon
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PC-APRV
80
FiO₂
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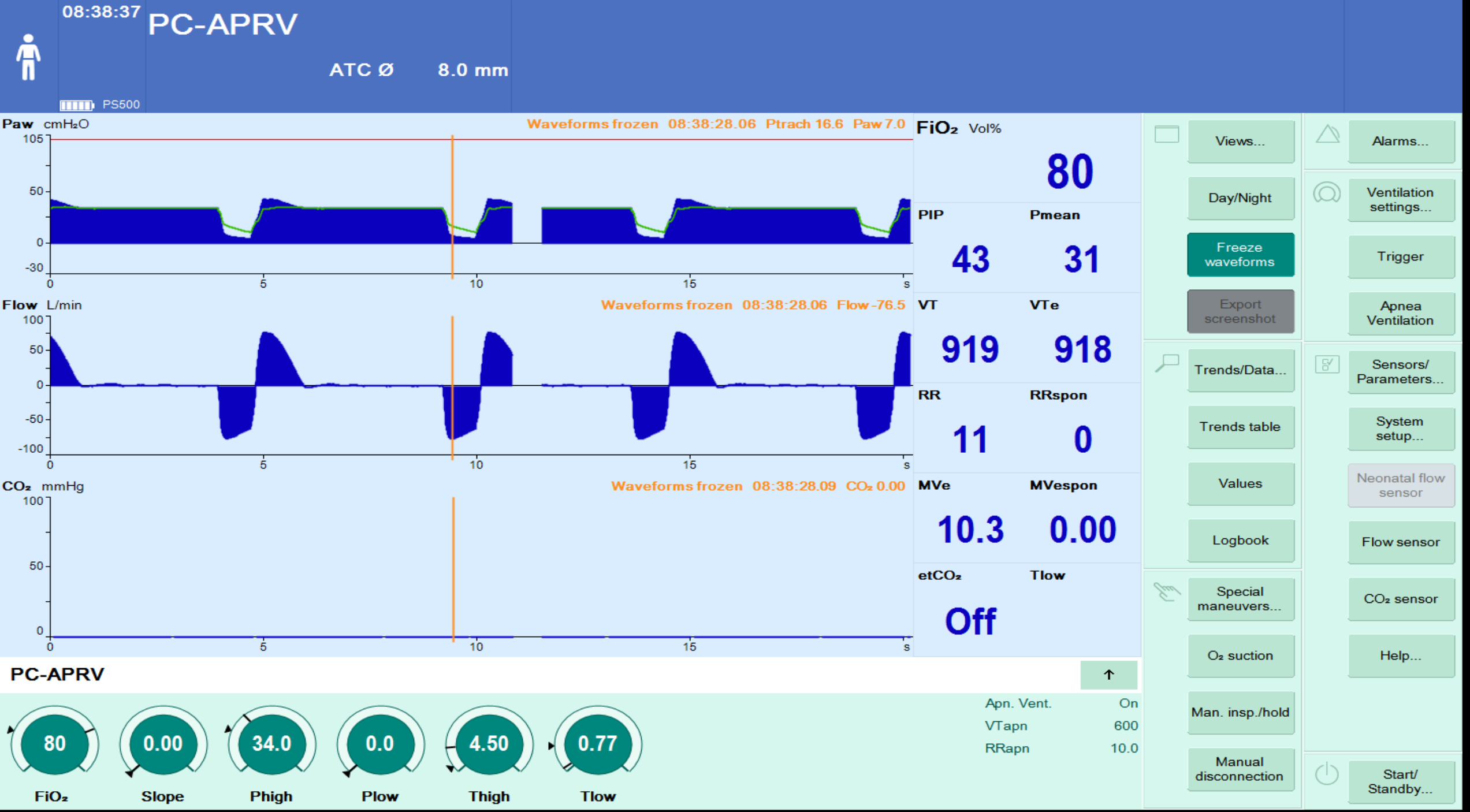
VTapn 600
RRapn 10.0


08:38:37
PC-APRV
ATC Ø 8.0 mm
PS500
Paw cmH₂O
Waveforms frozen 08:38:28.06 Ptrach 16.6 Paw 7.0
105
50
0
-30
0
5
10
15
s
Flow L/min
Waveforms frozen 08:38:28.06 Flow -76.5
100
50
0
-50
-100
CO₂ mmHg
Waveforms frozen 08:38:28.09 CO₂ 0.00
FiO₂ Vol%
80
PIP
43
Pmean
31
VT
919
VTe
918
RR
11
RRspon
0
MVe
10.3
MVespon
0.00
etCO₂
Off
Tlow
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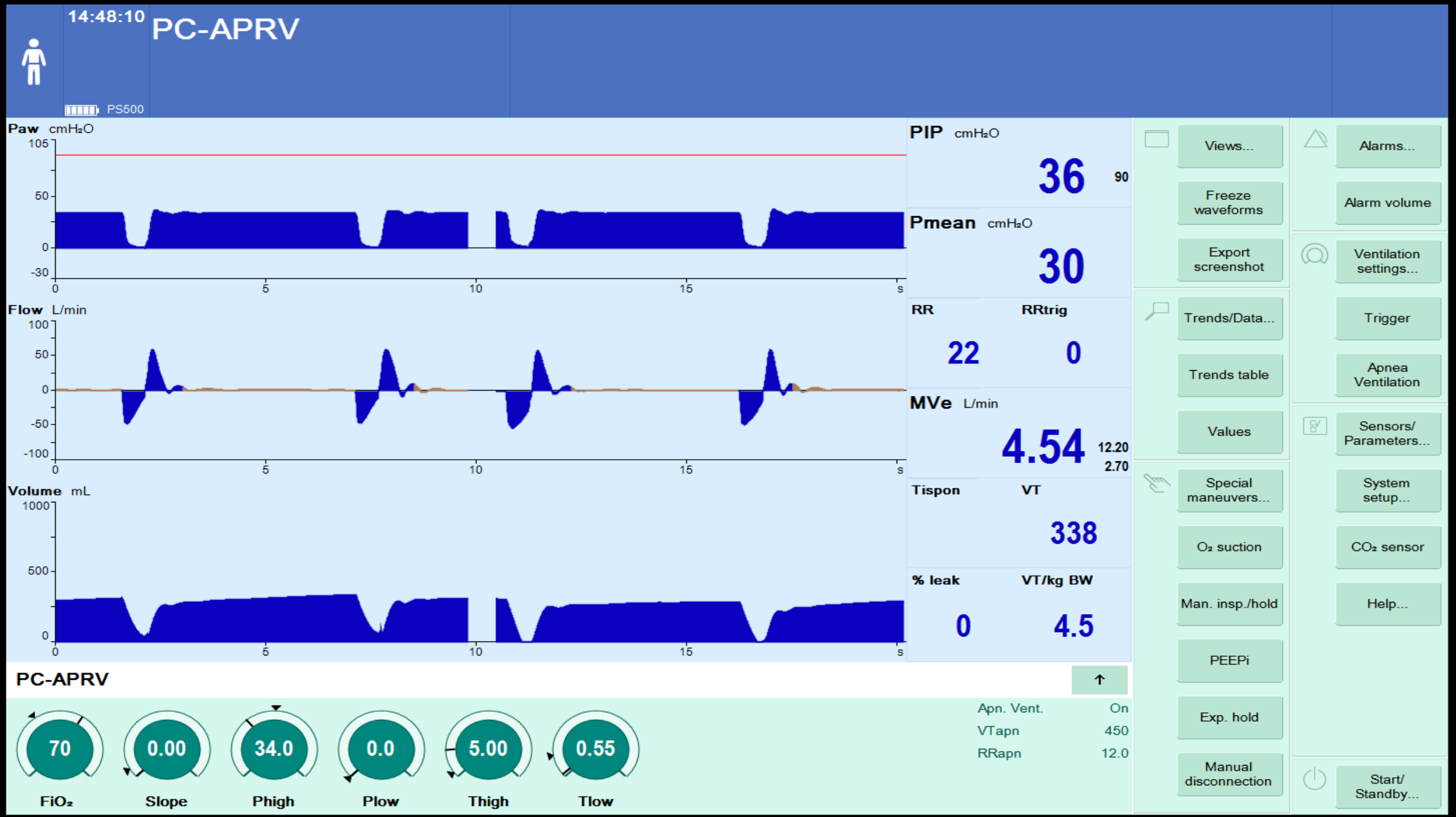
Slope
34.0
Phigh
0.0
Plow
4.50
Thigh
0.77
Tlow


14:48:10
PC-APRV
PS500
Paw cmH₂O
105
50
0
-30
0
5
10
15
s
Flow L/min
100
50
0
-50
-100
Volume mL
1000
500
PIP cmH₂O
36
90
Pmean cmH₂O
30
RR
RRtrig
22
0
MVe L/min
4.54
12.20
2.70
Tispon
VT
338
% leak
VT/kg BW
0
4.5
Views...
Freeze waveforms
Export screenshot
Trends/Data...
Trends table
Values
Special maneuvers...
O₂ suction
Man. insp./hold
PEEPi
Exp. hold
Manual disconnection
Alarms...
Alarm volume
Ventilation settings...
Trigger
Apnea Ventilation
Sensors/ Parameters...
System setup...
CO₂ sensor
Help...
Start/ Standby...
PC-APRV
70
FiO₂
0.00
Slope
34.0
Phigh
0.0
Plow
5.00
Thigh
0.55
Tlow
Apn. Vent. On
VTapn 450
RRapn 12.0

T Low

- Will vary if airway is changed and ventilator
- Laboratory data validates T Low set to terminate at 75% of peak expiratory flow rate
- Must measure per shift and with ventilator assessments
- **DO NOT** stretch T Low to increase minute volume

Original Investigation | ASSOCIATION OF VA SURGEONS

Mechanical Breath Profile of Airway Pressure Release Ventilation

The Effect on Alveolar Recruitment and Microstrain in Acute Lung Injury

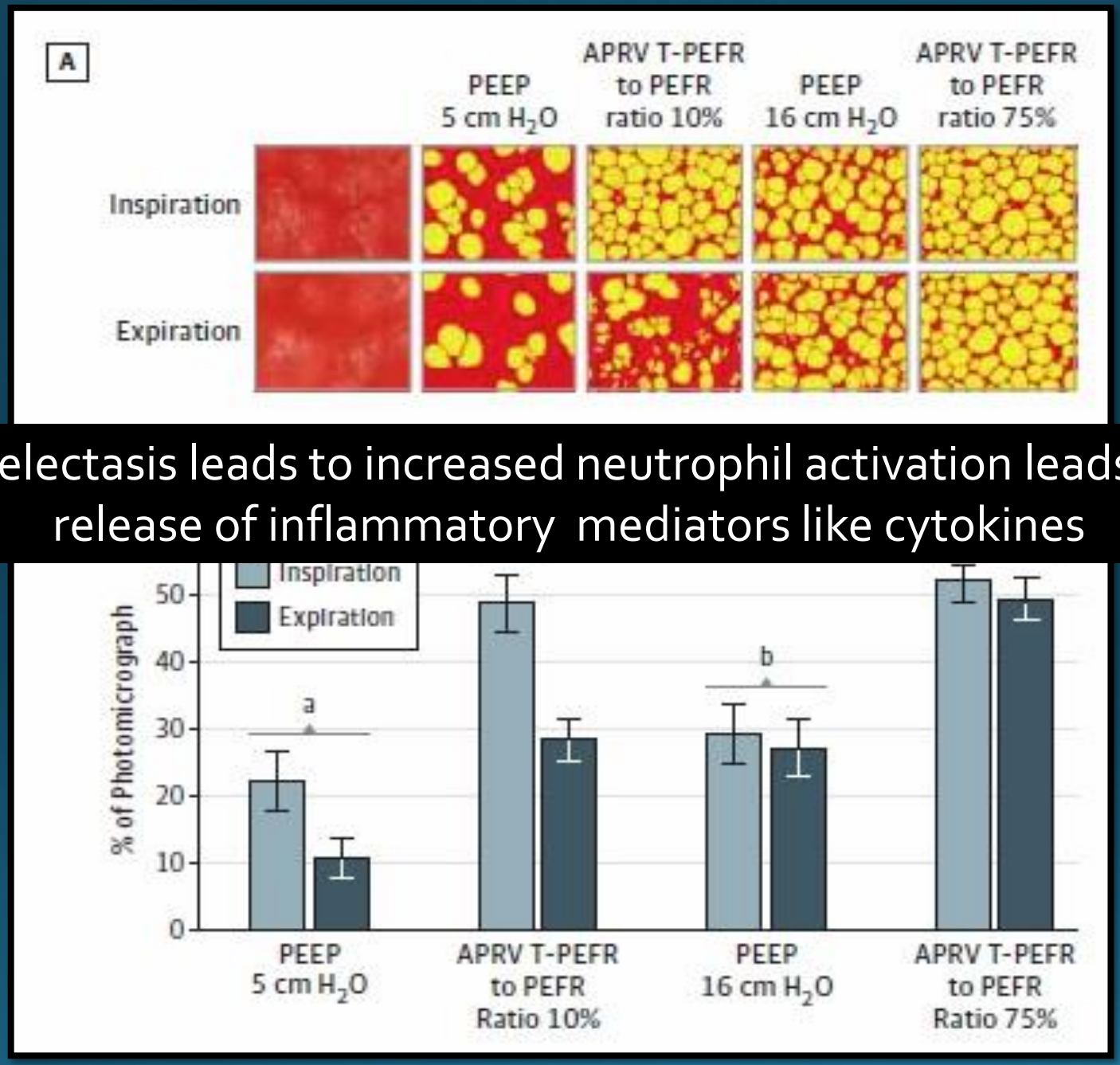
Michaela Kollisch-Singule, MD; Bryanna Emr, MD; Bradford Smith, PhD; Shreyas Roy, MD; Sumeet Jain, MD; Joshua Satalin, BS; Kathy Snyder; Penny Andrews, RN; Nader Habashi, MD; Jason Bates, PhD; William Marx, DO; Gary Nieman, BA; Louis A. Gatto, PhD

Male Sprague-Dawley rats
450-500 grams

ARDS net - CMV VT 6 ml/kg
PEEP – 5, 10, 16, 20 and 24 cmH_2O

APRV P High – 35 – 40 cmH_2O
T-PEFR/PEFR – 10, 25, 50 and 75%

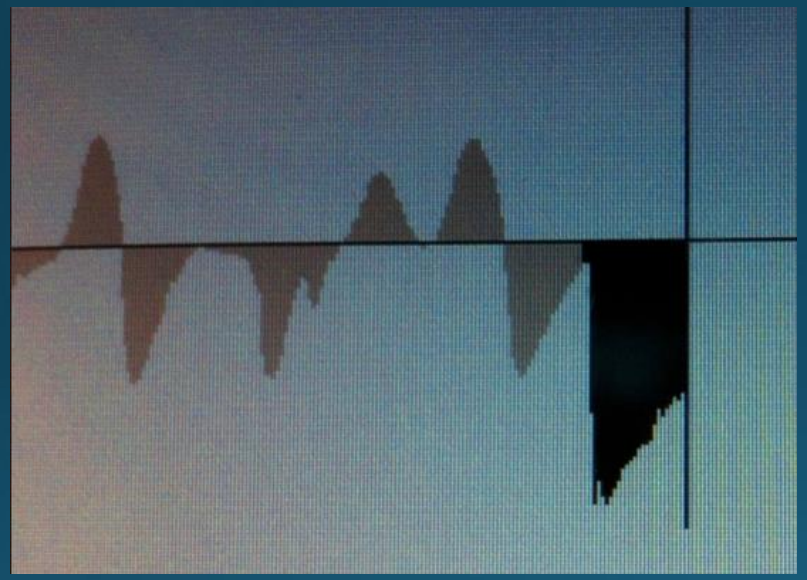
JAMA Surg. doi:10.1001/jamasurg.2014.1829
Published online September 17, 2014.

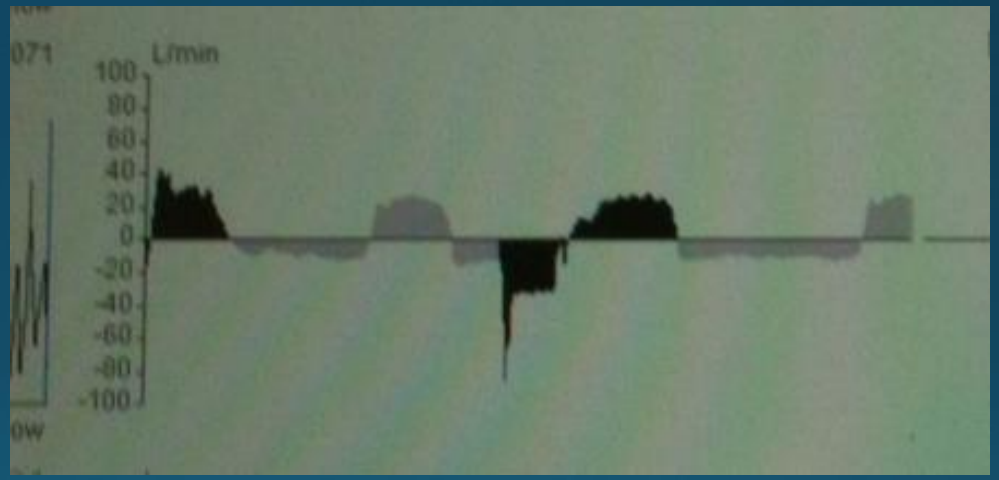


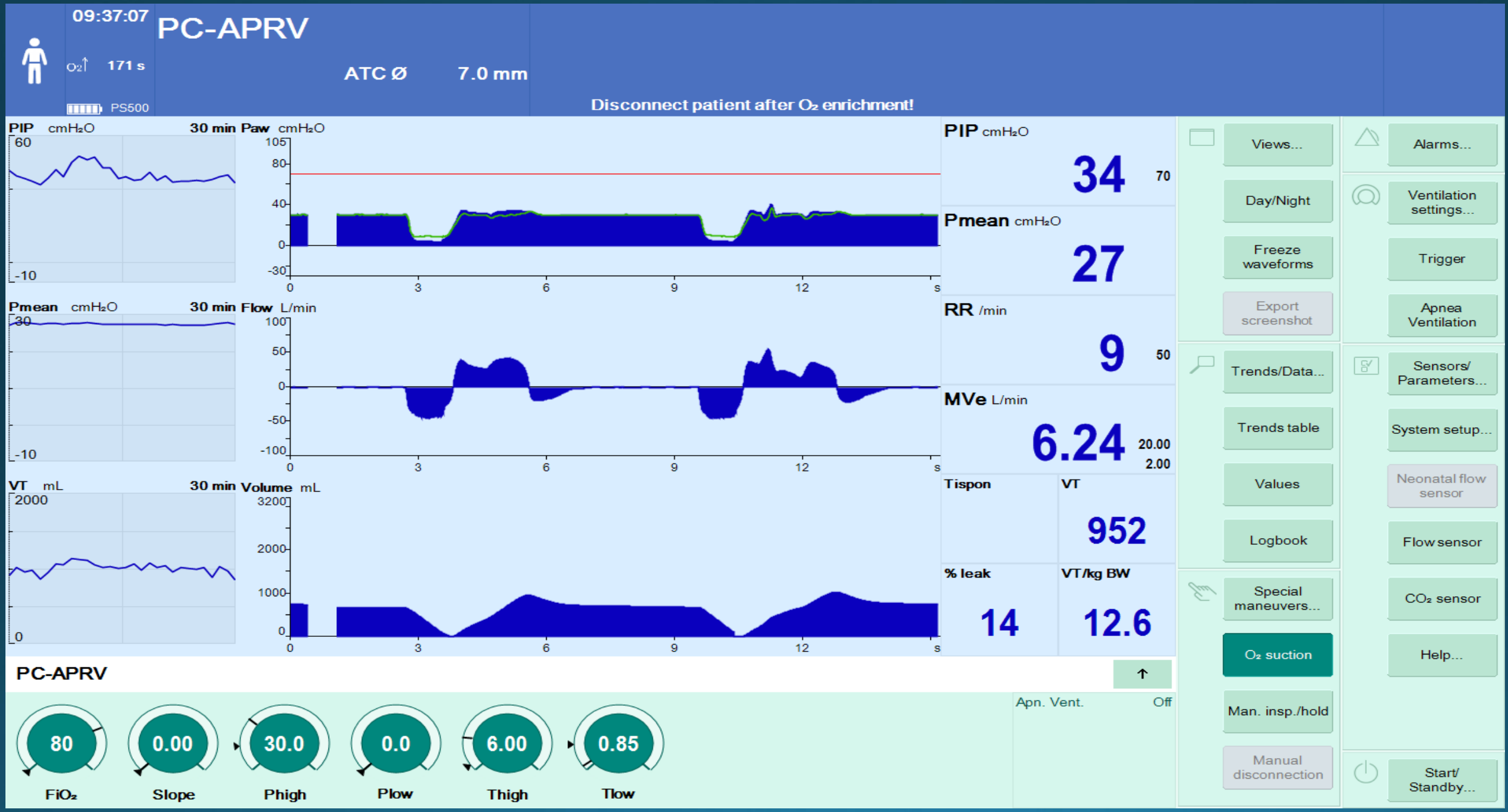
Atelectasis leads to increased neutrophil activation leads to release of inflammatory mediators like cytokines

JAMA Surg. doi:10.1001/jamasurg.2014.1829
Published online September 17, 2014.

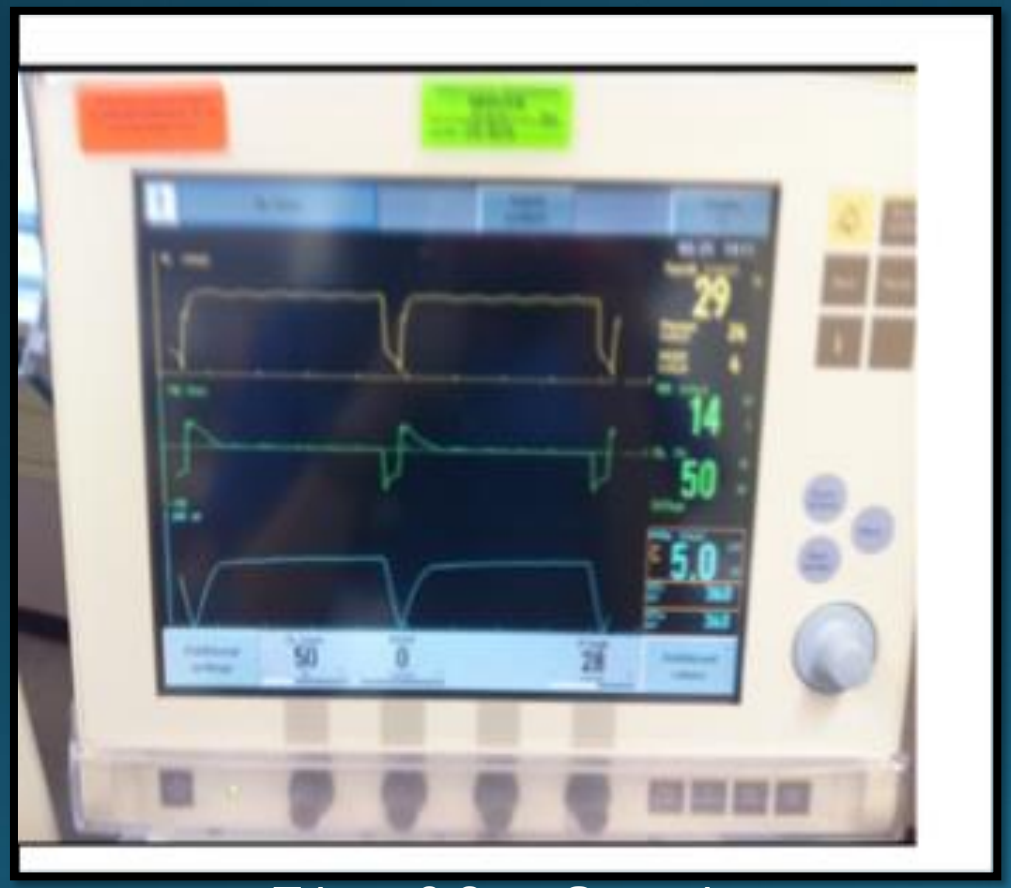
T Low



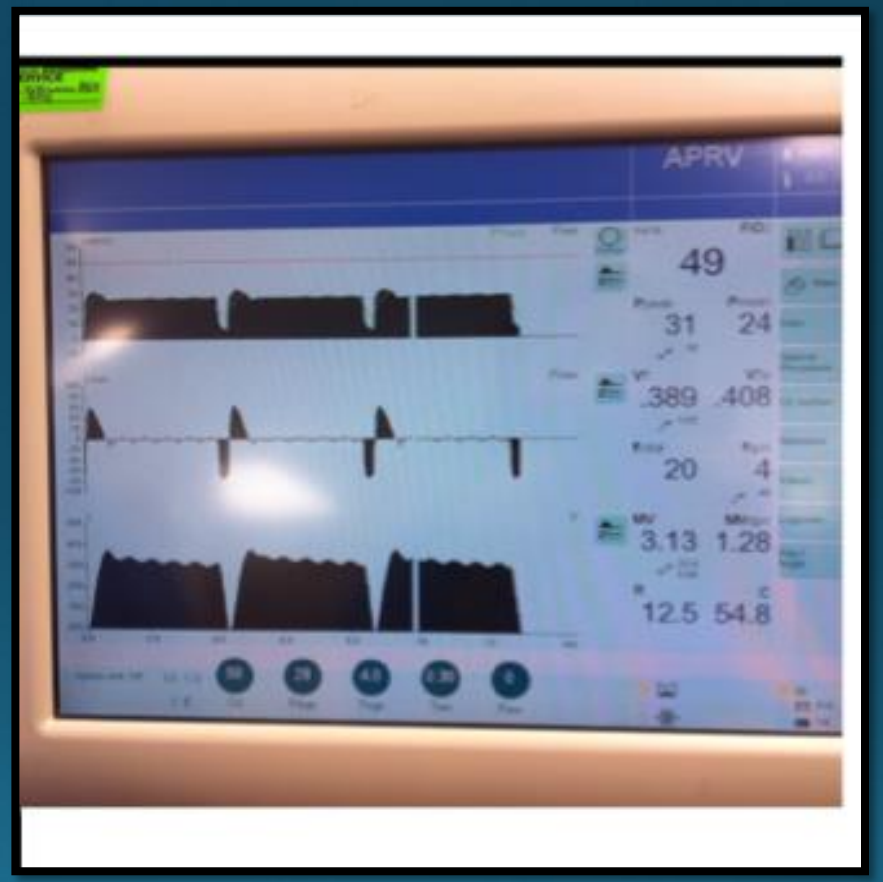



09:37:07
PC-APRV
O2↑ 171 s
ATC Ø 7.0 mm
PS500
Disconnect patient after O2 enrichment!
PIP cmH2O
30 min
Paw cmH2O
Pmean cmH2O
Flow L/min
VT mL
Volume mL
PIP cmH2O
34
70
Pmean cmH2O
27
RR /min
9
50
MVe L/min
6.24
20.00
2.00
Tispon
VT
952
% leak
14
VT/kg BW
12.6
Views...
Day/Night
Freeze waveforms
Export screenshot
Alarms...
Ventilation settings...
Trigger
Apnea Ventilation
Trends/Data...
Trends table
Values
Logbook
Sensors/ Parameters...
System setup...
Neonatal flow sensor
Flow sensor
CO2 sensor
Special maneuvers...
O2 suction
Man. insp./hold
Manual disconnection
Help...
Start/ Standby...
PC-APRV
Apn. Vent. Off
80
FiO2
0.00
Slope
30.0
Phigh
0.0
Plow
6.00
Thigh
0.85
Tlow

Always re-assess T Low



T Low 0.3 on Servo I



T Low 0.5 on Drager XL

Ventilator Assessment

TIME & SETTINGS	2000 18/0 10/0.5	2300 20/0 10/0.5
RR	6	9
VT	613	524
VE	3.7	3.1
T/P%	75%	60%

Ventilator Assessment

TIME & SETTINGS	2000 18/0 10/0.5	2300 18/0 10/0.5	0200 22/0 10/0.4	0300 25/0 10/.35
RR	6	9	12	10
VT	613	524	248	521
VE	3.7	3.1	2.94	2.86
T/P%	75%	60%		75%

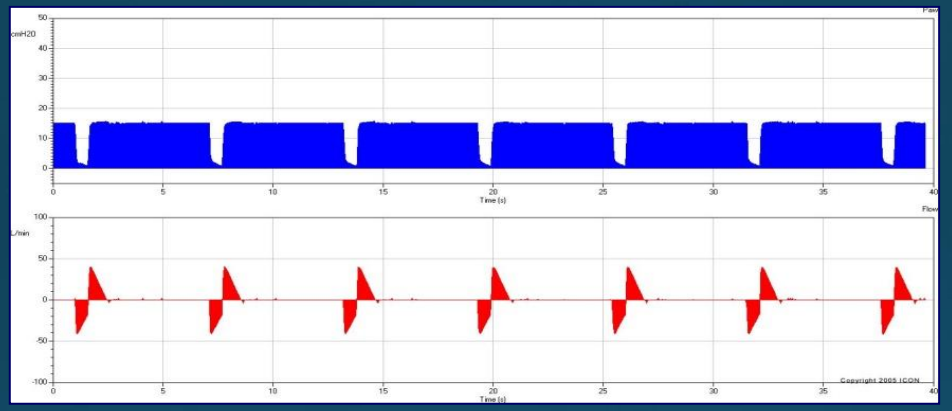
APRV & Spontaneous Breathing

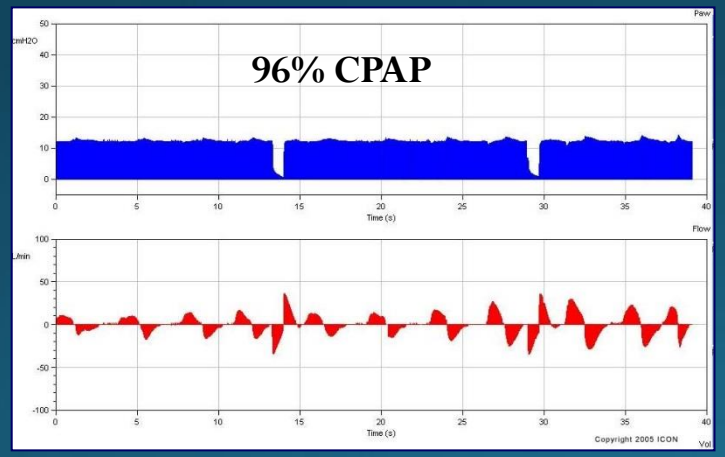
APRV can be used in patients who are not spontaneously breathing

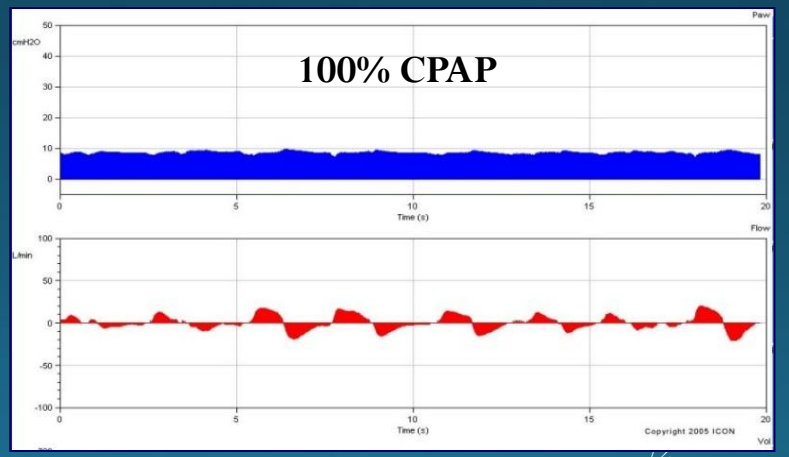
However, APRV enhances the benefits of spontaneous breathing

Bulk of APRV is CPAP

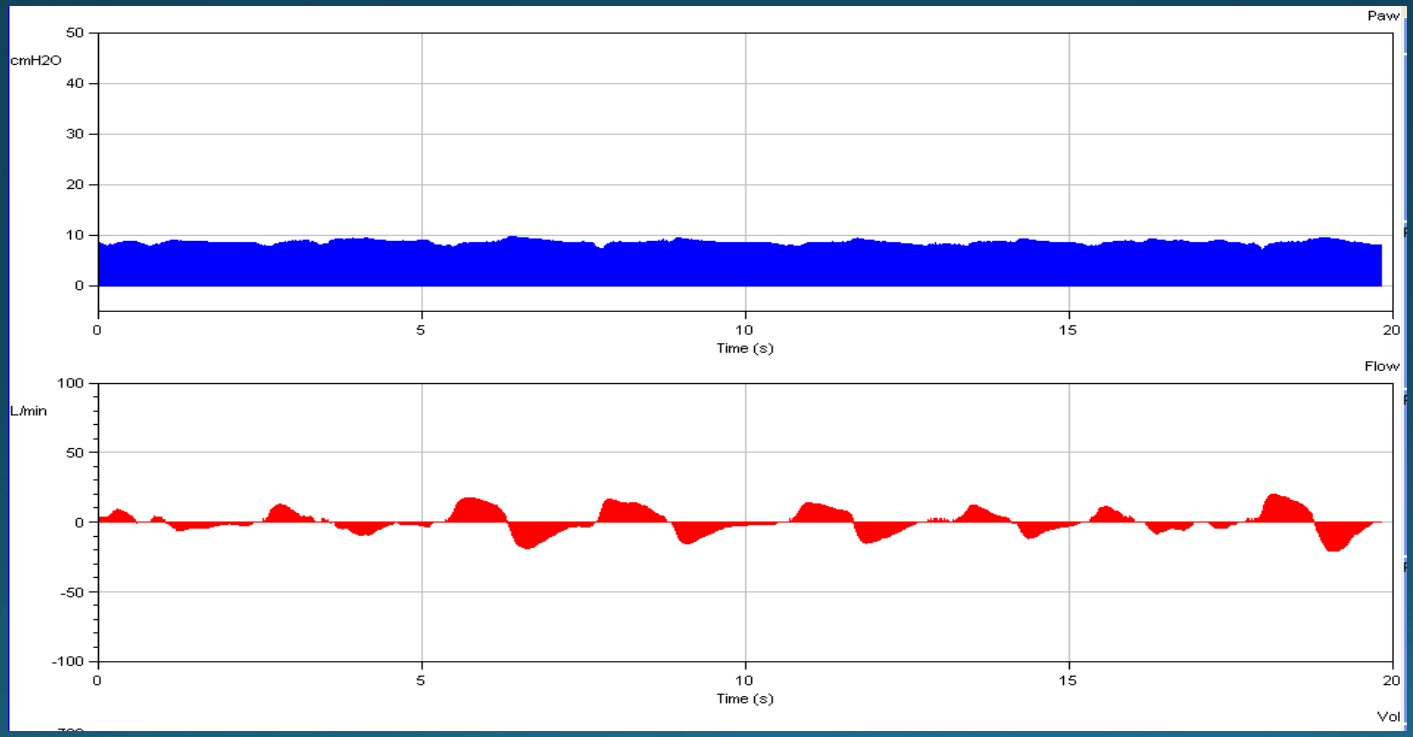
80-85 PEDIATRICS 90% ADULTS IN CPAP







APRV Now Becomes CPAP



CARDIO-Pulmonary Considerations

- Perfusion
- Volume resuscitation
  - The ventilator can't do it all!

Summary

- APRV may use the same inflating pressures as conventional, but with different timing.
- End expiratory lung volume is controlled and monitored with flow and time versus pressure.
- Spontaneous breathing is highly encouraged although **NOT** necessary.
- Literature exists to support APRV *preventing* ALI/ARDS.

APRV PROTOCOLS

WWW.APRVNETWORK.ORG


That's all Folks!

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