Journal of Mechanical Ventilation

Utilizing respiratory rate in APRV-TCAV protocol on Puritan Bennett ventilator. A case report

Jiaxiao Li, [1] Mohammed S O Al-Amoudi [2]

DOI: https://doi.org/10.53097/JMV10121

Cite: Li J, Al-Amoudi MSO. Utilizing Respiratory Rate in APRV-TCAV protocol on Puritan Bennett Ventilator. A Case Report. J Mech Vent 2025; 6(1):44-51.

Abstract

Background
Airway Pressure Release Ventilation (APRV), particularly with the Time-Controlled Adaptive Ventilation (TCAV) protocol, is known to improve oxygenation and respiratory mechanics. However, its role in managing refractory hypercapnia remains underexplored. This case report highlights APRV with TCAV as a potential strategy to tackle refractory hypercapnia.
Case Report
A 43-year-old woman with acute hypoxic and hypercapnic respiratory failure was admitted to our intensive care unit. Over the first 24 hours of management via conventional ventilation modes, she progressed to refractory hypercapnia, leading us to initiate modified APRV settings with TCAV protocol on the Puritan Bennett 980 ventilator (PB 980). This intervention led to rapid improvement in $PaCO_2$, successful transition to PSV, and eventual liberation.
Discussion
Our literature review revealed limited research on the use of higher controlled respiratory rates in APRV with TCAV. This case demonstrates the potential of this approach, emphasizing the importance of adhering to TCAV principles while optimizing respiratory rate settings. Additionally, we provide insights into APRV titration on the PB 980.
Conclusion
This report supports the use of APRV with higher controlled respiratory rates, adhering to TCAV protocols, as an effective strategy for managing refractory hypercapnia. Further research is warranted to establish evidence-based guidelines.

Keywords: CPAP, Time-Controlled Adaptive Ventilation (TCAV), Refractory Hypercapnia, Ventilation Strategies, Airway Pressure Release Ventilation (APRV)

Authors
1. RRT, Brockville General Hospital, Brockville, and department of surgery, Queen's University, Ontario, Canada
2. MD, FRCSC, FACS, Critical Care Brockville, and department of surgery, Queen's University, Ontario, Canada

Corresponding author: jiaxiaoli17@gmail.com
Conflict of interest/Disclosures: None

Introduction

Airway Pressure Release Ventilation (APRV) has been demonstrated to improve oxygenation and respiratory mechanics. [1] When utilized with the Time-Controlled Adaptive Ventilation (TCAV) protocol, it offers favorable hemodynamic profiles. [2] However, evidence on APRV with TCAV using a higher set respiratory rate to manage refractory hypercapnia remains limited. Here, we describe a 43-year-old woman with acute hypoxic and hypercapnic respiratory failure managed successfully with this approach.

Case Description

This is a 43-year-old woman with a history of bronchial asthma, morbid obesity, chronic kidney disease, and other comorbidities, presented to the emergency department with a four-day history of shortness of breath, increased respiratory distress, tachypnea, and oxygen saturation of 67% on room air. Examination revealed diffuse wheezing despite continuous nebulization with salbutamol and ipratropium bromide while on Non-Invasive Ventilation. Non-Invasive Ventilation therapy failed for refractory hypercapnia, and she was intubated and admitted to the ICU. Investigations: Her initial blood work is reflected in Table 1. Chest CT showed bilateral consolidation, multifocal opacities, and trace pleural effusion. Venous blood gas (VBG) analysis showed pH of 7.05, $PaCO_2$ of 58 mmHg post Non-Invasive Ventilation therapy, and bicarbonate of 17.6 mmol/L.

Interventions: Bronchoscopy was performed which showed no significant airway secretions. Continuous salbutamol nebulization, magnesium sulfate, methylprednisolone, and sodium bicarbonate infusion were initiated. Propofol and fentanyl achieved sedation targeting a Richmond Agitation-Sedation Scale (RASS) of -1 to -2.

Initially, the patient was placed on a PB 980 ventilator in assisted control/volume control plus (AC/VC+) mode, which is an adaptive ventilation mode rather than traditional volume controlled mode, targeting low tidal volumes (4-6 mL/kg ideal body weight) and plateau pressure (Pplat) <30 cmH_2O, peak pressures < 40 cm H_2O. The respiratory rate (RR) was adjusted to optimize minute ventilation while limiting auto-PEEP to < 2 cmH_2O. FiO_2 was titrated to maintain SpO_2 > 92%. To ensure measurement accuracy, Pplat was measured post intubation during paralytic state, which showed 30 cmH_2O, and static compliance (Cstat) was 11 mL/cmH_2O. Airway resistance was reflected by the difference between Ppeak and Pplat. Pplat was measured at 30 cmH_2O, and static compliance (Cstat) was 11 mL/cmH_2O. Lung volume recruitment maneuvers showed no improvement. Table 2A shows blood gas trends before APRV, day 1 of admission.

Overnight, ketamine was added for its bronchodilator effect, but transitioning from propofol to ketamine alone caused agitation and ventilator asynchrony. Propofol was reintroduced but asynchrony could not be resolved. Attempts at pressure support ventilation (PSV) were unsuccessful. The Minute Ventilation after optimizing the settings before APRV initiation was 5.7L, with Ppeak of 32 cm H_2O. APRV was subsequently initiated with TCAV protocols with the following settings:

- P-high: 30 cmH_2O
- P-low: 0 cmH_2O
- T-high: 2.18 seconds
- T-low: 0.55 seconds
- Respiratory Rate (RR): 22 breaths/min

These settings achieved a minute ventilation (MV) of 7.2 L and a tidal volume of ~330 mL per breath release. The patient, sedated only with ketamine at the time, had no spontaneous breathing. Fentanyl and propofol had been discontinued 4 and 6 hours earlier, respectively. Settings were titrated per TCAV protocol every two hours, reaching a maximum controlled RR of 24. She exhibited intermittent spontaneous respiration of up to 3 breaths/minute. Blood gas analysis is listed in Table 3A.

Overnight adjustments per TCAV protocol included titrating T-low to maintain an end-exhalation flow rate ≥75% of the peak expiratory flow rate. By morning, APRV settings were adjusted to a P-high of 26 cmH_2O, RR of 20, T-high of 2.7 seconds, and T-low of 0.3 seconds.

As the patient's condition improved, sedation was adjusted with the reintroduction of propofol for better comfort. Salbutamol nebulization was switched to salbutamol/ipratropium every 2 hours, and sodium bicarbonate infusion was stopped. P-high was weaned to target 6–8 ml/kg IBW before reducing RR. By the end of the day, APRV settings were P-high 16 cmH_2O, P-low 0 cmH_2O, RR 10, T-high 5.6 seconds, T-low 0.4 seconds, PS 26 cmH_2O (actual PS was 10 cmH_2O, discussed later). These settings yielded an MV of ~8.5L, total RR of 24, and Vt ~250ml per controlled breath drop. After assessing sufficient spontaneous breathing, the patient was transitioned to Pressure Support mode (PSV) with PEEP 11 cmH_2O, PS 12 cmH_2O, and FiO_2 0.45. Blood gas analysis are listed in Table 3B.

She was transitioned to PSV, then extubated to Non Invasive Ventilation (NIV), and finally to nasal cannula. The patient

expressed gratitude, emphasizing her preference to avoid future intubation.

Table 1: Initial laboratory values.

Venous Blood Gas pH	7.22	**Platelet**	128 x 10^9/L	**Sodium**	136 mmol/L	**Creatinine**	436 µmol/L
$PvCO_2$	49 mmHg	**Neutrophil**	8.83 x 10^9/L	**Potassium**	3.2 mmol/L	**eGFR**	<15 mL/min/1.73m^2
HCO_3	20.1 mmHg	**Lymphocyte**	0.74 x10^9/L	**Chloride**	104 mmol/L	**Total Bilirubin**	5 µmol/L
Lactate	1.1 mmol/L	**Eosinophil**	0 x 10^9/L	**Anion Gap**	15	**Albumin**	36 g/L
WBC	10.67 x 10^9/L	**Protein**	97 g/L	**Magnesium**	0.72 mmol/L	**ALP**	12 U/L
Hbg	108 g/L	**Glucose**	6.4 mmol/L	**Calcium**	1.77 mmol/L	**ALT**	9 U/L
Hct	0.332 L/L	**Troponin**	38 µg/L	**Phosphate**	1.36 mmol/L		

Table 2A: Blood Gas Trends Before APRV, Day 1 of Admission.

Time	pH	$PaCO_2$ (mmHg)	HCO_3 (mmHg)	PaO_2 (mmHg)	FiO_2	Note
0757	7.15	53	18.5	66	0.65	After one hour in the ICU
1017	7.16	57	20.3	85	1.0	Pre-bronchoscopy. FiO_2 was increased to 1.0 for preoxygenation for bronchoscopy
1512	7.14	66	22.5	78	0.8	Post-bronchoscopy. Ppeak = 34 cmH_2O, Pplat = 30 cmH_2O. FiO_2 titrated to 0.8 to maintain SpO_2 > 92%
1759	7.20	65	25.4	72	0.8	
2054	7.17	68	24.8	85	0.75	

Table 2B: Blood Gas Trends Before APRV, Day 2 of Admission

Time	pH	$PaCO_2$ (mmHg)	HCO_3 (mmHg)	PaO_2 (mmHg)	Notes
0046	7.21	67	26.8		AC/VC+, RR controlled at 28
0241	7.22	67	27.4		PSV (initiated at 0150), PS 18 cmH_2O, PEEP 8 cmH_2O, total RR of 12
0538	7.21	71	28.4		PSV, PS 18 cmH_2O, PEEP 8 cmH_2O, total RR of 13. Placed back on AC/VC+ due to worsening blood gas result
1011	7.20	76	29.7		Propofol was stopped two hours prior. Patient switched back to PSV two hours prior to this blood gas. PS 18 cmH_2O, PEEP 8 cmH_2O, total RR of 12. PEEP increased, fentanyl stopped.
1455	7.23	74	31	67	PSV, PS 18 cmH_2O, PEEP of 12 cmH_2O, total RR of 15, FiO_2 0.5. APRV initiated

Table 3A: Blood Gas Trends on APRV, Day 2 of Admission

Time	pH	$PaCO_2$ (mmHg)	HCO_3 (mmHg)	Notes
1707	7.28	65	30.5	This is after 90 minutes of being on APRV. Titrated settings to TCAV protocol, RR increased to 24
2256	7.29	66	31.7	After 90 min of being on titrated settings.

Table 3B: Blood Gas Trends on APRV, Day 3 of Admission

Time	pH	$PaCO_2$ (mmHg)	HCO_3 (mmHg)	Notes
0205	7.32	65	33.5	
0539	7.30	69	34	
0941	7.30	67	33	
1424	7.33	64	33.7	
1730	7.33	63	33.2	P-high 16 cmH_2O, P-low 0 cmH_2O, RR 10, I:E 14:1
2159	7.34	63	34	PS 12 cmH_2O PEEP 15 cmH_2O
0208	7.39	58	35.1	PS 12 cmH_2O PEEP 13 cmH_2O

Literature Review and Discussion

Application of TCAV in Hypercapnia

The TCAV protocol, pioneered by Dr. Habashi, [3] emphasizes personalizing T-low during the expiratory phase to match individual lung mechanics, allowing the majority of the respiratory cycles to stay at the mandatory inspiratory pressure phases. The most up-to-date published review on TCAV explicitly involves adjusting expiratory time to maintain end-exhalation flow at ≥ 75% of the peak expiratory flow rate. This allows prevention of complete exhalation, promotes lung recruitment through auto-PEEP, mitigates volutrauma, and ensures effective CO_2 elimination during the "release" phases. [4] TCAV also facilitates spontaneous breathing, reducing patient-ventilator asynchrony, and employs a prolonged I:E ratio for personalized tidal volumes. [4,5] Consistent with Jain et al.'s emphasis on personalized T-low settings, [1] we strictly adhered to our patient's lung mechanics, ensuring appropriate flow rates to prevent ventilator induced lung injury while optimizing recruitment.

Despite its potential, the efficacy of true TCAV approach with strict protocol adherence remains a research gap. We conducted a PubMed and Cochrane library search with text word "TCAV" or "Time Controlled Adaptive Ventilation" provided several reviews outlining potential physiological benefits when TCAV is applied alongside APRV, only one meta-analysis in humans was identified, which provided inconclusive evidence regarding its advantages, alongside one ongoing pilot study in France. [6]

This systematic review and preplanned meta-analysis by Katzenschlager et al., failed to identify clinical trials that demonstrated the superiority of APRV with strict adherence to TCAV over other ventilation modes. Notably, Katzenschlager et al. highlighted a tendency to extend the release phase in randomized controlled trials on hypercapnia, rather than strictly titrating the respiratory rate per TCAV principles. [7,8]

Our application of TCAV in this case study demonstrates practical insights into addressing this research gap. To address refractory hypercapnia and acidosis after the failure of other ventilation strategies, we implemented APRV with strict TCAV principles, meanwhile increased respiratory rate to 24 breaths per minute to augment $PaCO_2$ clearance by creating more frequent release phases, with T-low settings adjusted every two hours based on real-time monitoring of end-expiratory flow to match evolving lung mechanics. While the calculated mean $PaCO_2$ levels before and after APRV initiation were similar (Mean $PaCO_2$ before APRV initiation was 66.4, mean $PaCO_2$ post APRV initiation was 64.4), the overall clinical trend was not reflected as the $PaCO_2$ level was trending upwards with worsening acidemia. Although permissive hypercapnia is an accepted strategy to minimize ventilator-induced lung injury (VILI), its tolerance depends on the clinical context. In this case, a pH of 7.23 with a $PaCO_2$ of 74 mmHg, if worsening, could pose risks beyond ventilator mechanics. The fact that $PaCO_2$ was stabilized after APRV suggests that the intervention helped prevent further deterioration and demonstrated its potential role in ventilatory management.

Though it is not recommended adding pressure support (PS) during APRV with TCAV protocol, as it may counteract the intended recruitment effects of spontaneous breathing, its use in this case was for assisting the patient in increasing minute ventilation during emergence from sedation and evolving respiratory efforts.

While APRV has demonstrated improved gas exchange compared to other ventilation modes under similar minute ventilation, [9,10] studies have yet to explore the potential of increasing respiratory rates (release phases) to enhance $PaCO_2$ clearance. Despite limited studies on increasing respiratory rates in TCAV for $PaCO_2$ clearance, our search ('APRV' and 'Hypercapnia') revealed only 13 results on PubMed and two Cochrane trials, none offering definitive guidance. Jain et al.'s review (1987–2016) noted just three human studies incorporating higher respiratory rates, [1] and subsequent research since 2017 has included only a few cases with higher controlled rates. Furthermore, these studies often lacked methodological clarity or deviated from TCAV principles.

Our findings align with emerging evidence suggesting that higher respiratory rates in TCAV could be a valuable strategy for managing refractory hypercapnia. Table 4 summarizes the variability and limitations of existing studies, showing the need for further investigation in this area.

Table 4: Overview of Studies on APRV and Higher Controlled Respiratory Rate Organized by Publication Year

Study	Population	RR (bpm)	Key Findings	Limitations
Räsänen et al. (1991) [11]	Adults	20	Higher RR used.	TCAV adherence unclear.
Sydow et al. (1994) [12]	Adults	24	Higher RR applied; unclear TCAV adherence.	Combined spontaneous and set RR not distinguished.
Yoshida et al. (2009) [13]	Adults	Not controlled	Spontaneous respiration improved cardiac index and venous return compared to PSV.	Respiratory rate is not controlled.
Kamath et al. (2010) [14]	Pediatrics	20	Pediatric-specific RR; spontaneous contributions unclear.	Applicability to adults limited.
Arshad et al. (2016) [15]	Case report	Not recorded	APRV corrected hypercapnia but deviated from TCAV (I:E = 1:4).	Controlled RR, inconsistent with TCAV protocol.
Miller et al. (2017) [16]	Survey	20-30 (8%)	Majority of clinicians opted for a lower set RR; 8% used 20-30 bpm.	Survey-based data, not clinical outcomes.
Kreyer et al. (2021) [17]	Adults	35	Applied T-high : T-low of 1:2 to manage hypercapnia.	Controlled RR Inconsistent with TCAV protocol.
Rola et al. (2022) [18]	Theoretical concept	~25	Hypothesized approach to approximate TCAV for patient transfer under sedation with pressure control mode.	Theoretical; not validated in clinical practice.
Simón et al. (2023) [19]	Adults	29.35	Observational study comparing APRV and PSV; promoted spontaneous ventilation.	Controlled RR not recorded.

While the increase in RR helped clear $PaCO_2$ in this case, it is important to recognize that a higher RR also increases mechanical power, linking to VILI. Raising RR without considering alveolar ventilation, dead space, and the risk of auto-PEEP may be harmful. Clinicians should be mindful of these factors when adjusting ventilator settings.

In addition, while TCAV is designed to improve lung recruitment and protection, some aspects of its implementation remain unclear. The recommended end-exhalation flow rate adjusted to 75% of peak exhalation flow rate criteria for setting T-low does not have a fully established physiological basis. Other methods such as setting the T-low according to the expiratory time constant may also be feasible. [20] More research is needed to establish the best method.

With increased RR, clinicians should also pay close attention to hemodynamic changes during titration as raising RR poses patients to increased risk of obstructive shock due to increased

total PEEP. An expiratory hold maneuver is commonly performed for measuring total PEEP, which may not be feasible for some ventilators.

Ventilators such as Getinge Servo and Hamilton ventilators allow for an expiratory hold maneuver, whereas others, like PB ventilators, do not. Drager ventilators offer an expiratory hold function, but the maneuver is often prolonged, increasing the risk of derecruitment. These differences in ventilator functionality should be considered when applying APRV. Tracking the trend of static compliance through measurement of driving pressure is also valuable. However, keep in mind that the accuracy in spontaneously breathing patients is questionable as spontaneous respiration alters accuracy of measurement.

In such cases, estimating driving pressure by incorporating total PEEP measurements may provide a more practical approach.

Transitioning from Deep Sedation with APRV

APRV supports $PaCO_2$ clearance by integrating spontaneous breathing with a controlled respiratory rate, making it effective for patients emerging from deep sedation. Initially, our patient's spontaneous breathing was insufficient. Putensen et al. showed that spontaneous breathing improves V/Q distribution in ARDS, but APRV without spontaneous breathing is similar to PCV. [21] By employing a higher controlled respiratory rate, we achieved effective gas exchange while supporting the gradual recovery of spontaneous breathing and enhancing V/Q distribution. Effective sedation is crucial during APRV to prevent agitation, which can impair gas exchange and exacerbate bronchoconstriction.

Zhou et al. emphasized balancing sedation with spontaneous breathing, [22] highlighting the importance of individualized sedation strategies, especially in patients with complex comorbidities. [23]

Technical Considerations on PB 980 Ventilators

When titrating T-low on the PB 980 ventilator, the peak point on the exhalation flow waveform should not be used as the peak exhalation flow rate. Instead, the second-highest flow rate, which descends smoothly during exhalation, represents the true peak exhalation flow rate. This distinction arises because the ventilator's exhalation valve, defined as controlling PEEP, [24] causes the initial peak in the waveform when transitioning from P-high to P-low, reflecting valve mechanics rather than lung exhalation. In addition, the PS setting should be set as Ppeak, meaning the actual PS equals the gradient between Ppeak and P-high, allowing PS breaths to be delivered (Figure 1A and Figure 1B).

What's more, spontaneous breaths taken at the end of P-high may prolong T-low due to synchronization settings potentially altering the intended lung protection strategy. These variations emphasize the need for careful adjustments when applying TCAV.

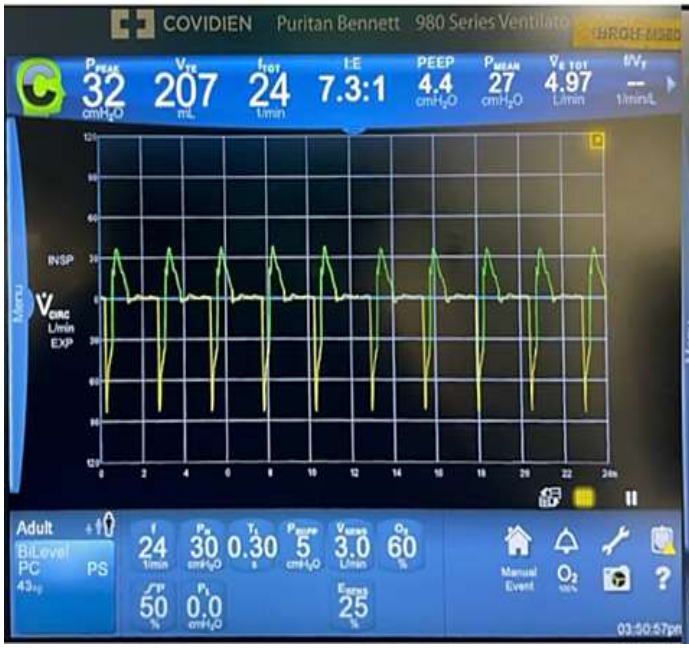

Figure 1A

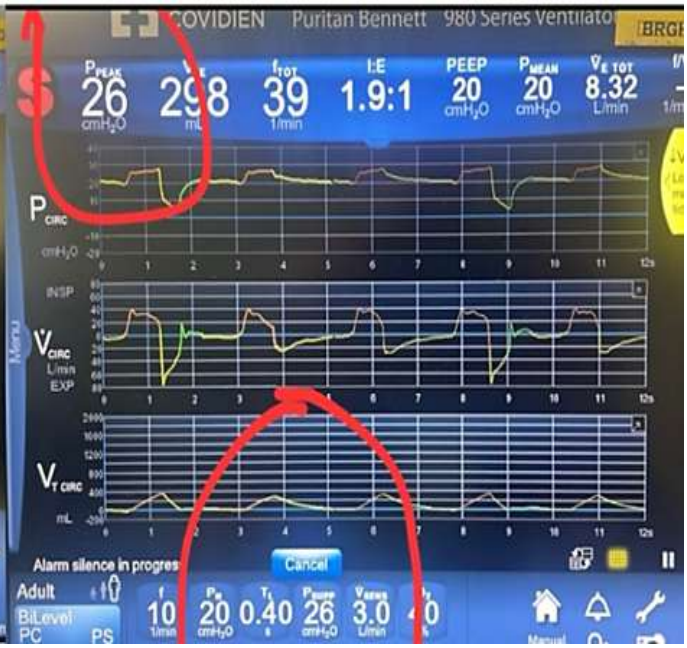

Figure 1B

Figure 1A showed the distinction between valve mechanism and actual exhalation phases. Figure 1B showed the relationship between PS setting and Ppeak.

Conclusion

APRV with TCAV protocol, incorporating a higher controlled respiratory rate, successfully managed refractory hypercapnia in this case. Clinicians should consider tailoring APRV settings to individual lung mechanics and leveraging higher respiratory rate when conventional strategies fail. Further research is warranted to establish evidence-based guidelines for this approach.

References

1. Jain SV, Kollisch-Singule M, Sadowitz B, et al. The 30-year evolution of airway pressure release ventilation (APRV). Intensive Care Med Exp 2016; 4(1):11.

2. Al-Khalisy H, Nieman GF, Kollisch-Singule M, et al. Time-Controlled Adaptive Ventilation (TCAV): a personalized strategy for lung protection. Respir Res 2024; 25(1):37.

3. Habashi NM. Other approaches to open-lung ventilation: airway pressure release ventilation. Crit Care Med 2005; 33(3 Suppl):S228-S240.

4. Al-Khalisy H, Nieman GF, Kollisch-Singule M, et al. Time-Controlled Adaptive Ventilation (TCAV): a personalized strategy for lung protection. Respir Res 2024; 25(1):37.

5. Spiegel R, Hockstein M. Airway Pressure Release Ventilation: A field guide for the emergency physician. Emerg Med Clin North Am. 2022;40(3):489-501. doi:10.1016/j.emc.2022.05.004

6. Pequignot B. Effects of TCAV and Volume Control Ventilation on the distribution of aerated lung parenchyma in ARDS patients (TCAV-CT). Clinicaltrials.gov. February 7, 2024. Accessed December 16, 2024. https://clinicaltrials.gov/study/NCT05874973.

7. Katzenschlager S, Simon CM, Rehn P, et al. Time-controlled adaptive ventilation in patients with ARDS-lack of protocol adherence: a systematic review. Crit Care 2023; 27(1):57.

8. Ibarra-Estrada MÁ, García-Salas Y, Mireles-Cabodevila E, et al. Use of Airway Pressure Release Ventilation in patients with acute respiratory failure due to COVID-19: Results of a single-center randomized controlled trial. Crit Care Med 2022; 50(4):586-594.

9. Stock MC, Downs JB, Frolicher DA. Airway pressure release ventilation. Crit Care Med 1987; 15(5):462-466.

10. Maung AA, Luckianow G, Kaplan LJ. Lessons learned from airway pressure release ventilation. J Trauma Acute Care Surg 2012; 72(3):624-628.

11. Räsänen J, Cane RD, Downs JB, et al. Airway pressure release ventilation during acute lung injury: a prospective multicenter trial. Crit Care Med 1991; 19(10):1234-1241.

12. Sydow M, Burchardi H, Ephraim E, et al. Long-term effects of two different ventilatory modes on oxygenation in acute lung injury. Comparison of airway pressure release ventilation and volume-controlled inverse ratio ventilation. Am J Respir Crit Care Med 1994; 149(6):1550-1556.

13. Yoshida T, Rinka H, Kaji A, et al. The impact of spontaneous ventilation on distribution of lung aeration in patients with acute respiratory distress syndrome: airway pressure release ventilation versus pressure support ventilation. Anesth Analg 2009; 109(6):1892-1900.

14. Kamath SS, Super DM, Mhanna MJ. Effects of airway pressure release ventilation on blood pressure and urine output in children. Pediatr Pulmonol 2010; 45(1):48-54.

15. Arshad Z, Prakash R, Aggarwal S, et al. Ventilating patient with refractory hypercarbia: Use of APRV mode. J Clin Diagn Res 2016; 10(1):UD01-UD2.

16. Miller AG, Gentile MA, Davies JD, et al. Clinical management strategies for airway pressure release ventilation: A survey of clinical practice. Respir Care 2017; 62(10):1264-1268.

17. Kreyer S, Baker WL, Scaravilli V, et al. Assessment of spontaneous breathing during pressure controlled ventilation with superimposed spontaneous breathing using respiratory flow signal analysis. J Clin Monit Comput 2021; 35(4):859-868.

18. Rola P, Daxon B. Airway pressure release ventilation with time-controlled adaptive ventilation (TCAV™) in COVID-19: A community hospital's experience. Front Physiol 2022; 13:787231.

19. Simón JMS, Montosa CJ, Carmona JFM, et al. Effects of three spontaneous ventilation modes on respiratory drive and muscle effort in COVID-19 pneumonia patients. BMC Pulm Med 2023 ;23(1):333.

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

21. 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(4 Pt 1):1241-1248.

22. Zhou Y, Jin X, Lv Y, et al. Early application of airway pressure release ventilation may reduce the duration of mechanical ventilation in acute respiratory distress syndrome. Intensive Care Med 2017; 43(11):1648-1659.

23. Cascella M, Bimonte S, Di Napoli R. Delayed emergence from anesthesia: What we know and how we act. Local Reg Anesth 2020; 13:195-206.

24. Puritan Bennett - 980 series ventilator Operator's Manual. Accessed November 26, 2024.https://asiapac.medtronic.com/content/dam/covidien/library/us/en/product/acute-care-ventilation/PuritanBennett980Ventilator_OperatorsManual_en_OUS_PT00094084A00.pdf.

SMV 2025

SOCIETY OF MECHANICAL VENTILATION CONFERENCE

EXPAND YOUR KNOWLEDGE, NETWORK, IMPROVE OUTCOME, HAVE FUN

Speakers

JOHN DOWNS MD
ANDREW FENG MD
BRENT MATSUDA MD
CHERIE CHU PHARMD
DAVID WILLMS MD
KIMIYO YAMASAKI RRT

BENJAMIN DAXON MD
RYOTA SATO MD
SAMUEL EVANS MD
STEPHEN TUNNEL RRT
EHAB DAOUD MD

DATE: JUNE 30 - JULY 1, 2025
7:00 AM TO 12: 30PM

LOCATION: KUAKINI MEDICAL CENTER AUDITORIUM
347 NORTH KUAKINI STREET, HONOLULU

IN PERSON OR ONLINE
REGISTER TODAY

https://societymechanicalventilation.org/2025-2/