

Airway Pressure Release Ventilation Benefits in a Patient with Chronic Lymphocytic Leukemia

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Abstract

Background: Airway pressure release ventilation (APRV) is a novel mode of mechanical ventilation (MV) used in the treatment of acute respiratory distress syndrome (ARDS) for patients with severe hypoxemia with a strategy to prevent ventilator-induced lung injury (VILI). APRV can avert VILI because it can limit alveolar-distending pressures. This is a case report of a 45-year-old man diagnosed with chronic lymphocytic leukemia who later developed community-acquired pneumonia and ARDS. **Methods:** APRV was applied successfully. Initially, he was unsuccessfully managed on conventional ventilation using an “open-lung” ventilation strategy (high positive end-expiratory pressure, high respiratory rate, and low tidal volume), recruitment maneuvers, and prone positioning. **Results:** A change in the ventilation mode to APRV resulted in the reduction of extravascular lung water as indicated by a chest X-ray, improvement in the oxygenation indices, and successful liberation from the ventilator. **Conclusion:** This case report concluded that APRV is safe in patients diagnosed with ARDS if other “open-lung” MV approaches and prone positioning have failed.

Keywords: Acute respiratory distress syndrome, airway pressure release ventilation, atelectrauma, barotrauma, chronic lymphocytic leukemia, lung protective strategies, mechanical ventilation, recruitment maneuvers, ventilator-induced lung injury, volutrauma

INTRODUCTION

Airway pressure release ventilation (APRV) has continued to gain momentum in intensive care settings as an alternative mechanical ventilatory management method for patients with acute respiratory distress syndrome (ARDS). APRV is a mode of ventilation consisting of continuous positive airway pressure ventilation with two levels of pressures so that patients can breathe spontaneously. The ventilation is pressure-limited and time-cycled. Two levels of pressure and time settings can be set: high pressure (P-high), low pressure (P-low), time high (T-high), and time low (T-low). The mode has the advantage of allowing mechanical ventilation (MV) patients to breathe spontaneously, thereby increasing pulmonary blood flow, improving oxygenation, and obtaining better gaseous exchange.^[1-4] Other ventilatory management methods, such as high-frequency oscillatory ventilation (HFOV) and “open-lung” ventilator strategies, may result in ventilator-induced lung injury (VILI), barotrauma, and volutrauma.^[5-7]

There is no reported experience of a case report of a patient diagnosed with chronic lymphocytic leukemia (CLL) with

respiratory failure who later developed community-acquired pneumonia (CAP) and ARDS that was managed with APRV. The patient was successfully managed on the MV using APRV and was extubated in the intensive care unit (ICU) at our hospital.

CASE REPORT

A 45-year-old male with CLL diagnosed 10 years prior presented at another hospital with fever, cold, shortness of breath, and cough. He was diagnosed with CAP needing MV. His medical history was not available because the patient had received MV at another hospital. The patient was transferred from one ICU to King Fahad Medical City’s ICU. The only history recorded in the patient’s chart was his intake of camel milk mixed with camel urine. Middle East respiratory

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syndrome coronavirus and influenza A tests were negative. The patient was on prophylaxis, meropenem, clarithromycin, and moxifloxacin for antibiotic treatment. In addition, he received Tamiflu as an antiviral treatment.

The patient was hemodynamically stable with a slight fever (axillary temperature recorded at 38.1°C); his septic screen (sputum and blood culture), chemistry, renal profile, and electrocardiogram were all within acceptable limits. Chest X-ray (CXR) for the prior 5 days were consistent with bilateral pulmonary infiltrates and air bronchograms [Figure 1]. His bilateral breath sounds were scattered crackles throughout

the lung field. The patient was sedated with 200 mcg/h intravenous (IV) of fentanyl and 5 mg IV of midazolam. His Glasgow Coma Scale was 11.

After 2 days of conventional ventilation, there were frequent desaturations, as recorded by pulse oximetry, which was as low as 70% with an increased fraction of inspired oxygen (FiO₂) reaching 1.0 on MV. By day 4, the team initiated 10 mcg/kg/min IV of cisatracurium in addition to 100 mcg/h IV of fentanyl and 3 mg IV of midazolam. The initiation of the sedation and paralysis was due to the patient having extreme agitation with patient-ventilator dyssynchrony as revealed by waveform analysis and increased work of breathing on MV. Initially, the patient was ventilated with tidal volume (VT) of 8 ml/kg of the ideal body weight (IBW) on controlled mandatory ventilation with AutoFlow (Drager Evita XL, Lubeck, Schleswig-Holstein, Germany). Positive end-expiratory pressure (PEEP) gradually increased to 14 cm H₂O with a plateau pressure (P_{PLT}) >30 cm H₂O.

By day 5, the team put the patient in a prone position due to refractory hypoxemia (partial pressure of oxygen in the blood [PaO₂] of 56–62 mmHg) that was not responding to PEEP of 14 cm H₂O and high FiO₂ of 1.0. To reduce VILI, the ARDSnet protocol was implemented. VT was adjusted to 5 ml/kg to maintain P_{PLT} below 30 cm H₂O, PEEP was increased to 18 cm H₂O, and the respiratory rate (RR) was increased to 32 breaths per minute (BPM) over a 2-day period.^[8] The team accepted arterial blood gas (ABG) of pH 7.25 or more, PaCO₂ of 60 mmHg or more, and SaO₂ of 88%–92% as a protective measure to reduce VILI. The following day, the patient was placed on a lung, recruitment maneuver (RM) with PEEP increased to 40 cm H₂O for 40 s and gradually reduced to 18 cm H₂O. The team continued placing the patient in the prone position twice daily for 8 h each time, once in

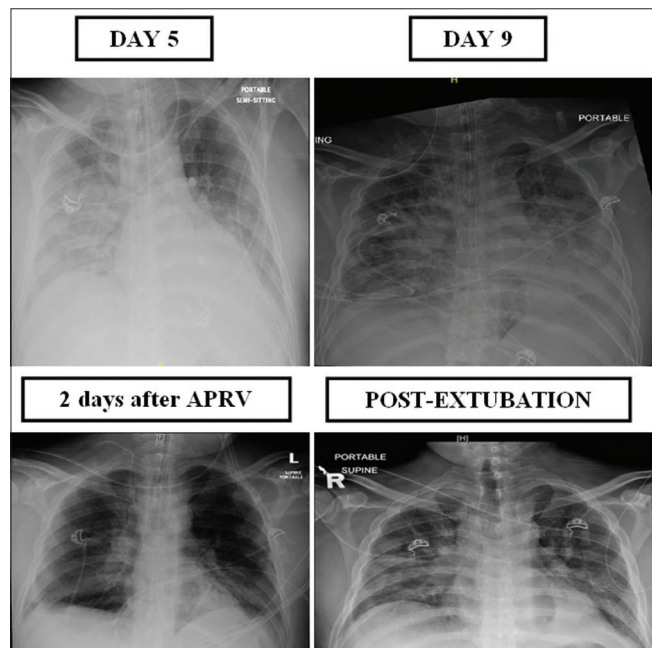


Figure 1: Chest X-ray

Table 1: Average per day ratio of partial pressure of oxygen in the blood to the fraction of inspired oxygen and oxygenation index (%) in conventional ventilation, airway pressure release ventilation, and pressure support ventilation modes

Day	CV		Day	APRV		Day	PSV	
	PaO ₂ /FiO ₂	OI (%)		PaO ₂ /FiO ₂	OI (%)		PaO ₂ /FiO ₂	OI (%)
1	213	7.1	10	136	23	12	200	7
2	101	13.9	11	212	12	13	158	9
3	84	23.2	12	236	9	14	215	5
4	81	25.8				15	246	4
5	105	22.9				16	303	2
6	90	27.8						
7	123	18.0						
8	128	16.0						
9	105	16.1						
10	104	19.2						

*PaO₂/FiO₂ ratio <100 is severe ARDS; PaO₂/FiO₂ ratio <200 is moderate ARDS; and PaO₂/FiO₂ ratio <300 is mild ARDS, **OI (%) <5 is good; OI (%) between 5 and 20 is bad; OI (%) >20 is worst. AFM: Aerosol face mask, NO: Nitric oxide, ARDS: Acute respiratory distress syndrome, CV: Conventional ventilation, APRV: Airway pressure release ventilation, PSV: Pressure support ventilation, PaO₂/FiO₂: Ratio of partial pressure of oxygen in the blood to the fraction of inspired oxygen, OI: Oxygenation index (Paw × FiO₂/PaO₂) 100 where Paw: Mean airway pressure, FiO₂: Fraction of inspired oxygen, PaO₂: Partial pressure of oxygen in the blood; days 5 to 10 proning was done twice daily (in the morning and at night); day 9 NO started; day 10 APRV started and proning stopped; day 11 NO discontinued; day 12 PSV started; day 16 patient extubated to AFM; days shown twice means there was a switch between modes

the morning and once at night. There was improvement in the saturation, which allowed for a reduction in FiO_2 from 1.0 to 0.60. PEEP was gradually decreased to 10 cm H_2O over a 4-day period [Appendix B for a slight improvement in oxygenation].

On the 9th day, the patient deteriorated with frequent desaturations in the low 80 s. The FiO_2 requirement increased from 0.40 to 0.80, and PEEP was increased to 14 cm H_2O despite the prone positioning [Table 1]. A CXR showed diffuse infiltration with air bronchograms and bibasilar effusions [Figure 1]. The team started nitric oxide (NO), a pulmonary vasodilator, at 20 parts per million because the $\text{PaO}_2/\text{FiO}_2$ ratio was in the low 100s [indicative of moderate-to-severe ARDS; Table 1] despite prone positioning twice daily to enhance oxygenation. The next day, the team initiated the patient on APRV in continuation with the NO and discontinued prone positioning to allow for better recruitment.

Within 2 days of APRV initiation, oxygenation improved allowing for a reduction of FiO_2 from 1.0 to 0.40 and the discontinuation of NO [Figure 1 and Table 1]. NO was discontinued on day 11 with FiO_2 limited to just 0.50. By day 12, sedation and paralytics were gradually weaned to allow for spontaneous respiration. Brain computed tomography (CT) and abdominal CT were unremarkable. The “drop and stretch” method was used by gradually dropping P-high and stretching T-high with the ABG within the acceptable limits. On day 3 of APRV initiation, the patient was switched to pressure support ventilation (PSV) to begin the process of weaning from the ventilator. His PSV level was gradually weaned from 14 cm H_2O to 5 cm H_2O and his PEEP level was gradually weaned from 10 cm H_2O to 5 cm H_2O as per APRV clinical protocol [Appendix]. By day 5 of PSV initiation, the patient was extubated to an aerosol face mask.

DISCUSSION

The basic premise of applying a mode of MV is to understand how it works and how to safely apply it to a critically ill patient. Therefore, the goal for all mechanically ventilated patients is to liberate them from the ventilator as soon as the original clinical problem requiring MV has been reversed. Daoud *et al.*^[9] described how clinicians interchangeably use APRV and bilevel positive airway pressure (BiPAP) to achieve the same results; however, the meanings and modes of applications are different. Daoud *et al.*^[9] described APRV as having an “extreme” inverse inspiratory to expiratory ratio while BiPAP does not. They contended that the problem of confusing APRV with BiPAP occurs for two reasons: (1) complexities that exist in the criteria used to define the modes and (2) different names purported by the ventilator manufacturers. They defined “extreme” as an inspiratory-to-expiratory ratio >2:1; nonetheless, this researcher disagrees with the term “extreme.” Anecdotal evidence suggests that this term has prevented some clinicians from adopting this mode of ventilation as an early intervention strategy to treat ARDS patients with

moderate-to-severe hypoxemia for fear of developing VILI. This ideology is contrary to the reason for initiating APRV in the first place. Indeed, further research must investigate the safety measures and advantages of using APRV to treat ARDS patients. The clinical strategy described by Daoud *et al.*^[9] in initiating APRV was the same strategy used in the case report [Appendix]. This strategy involves using P_{PLT} as the reference point for setting the P-high. This strategy of equating P_{PLT} to set the P-high is the most practical way to reduce VILI.

Based on this researcher’s experience, three important criteria allow for the successful application of APRV: early intervention, proper training of the respiratory critical care staff, and strict application of APRV clinical protocol [Appendix]. Chatburn *et al.*^[10] responded to Mireles-Cabodevila and Kacmarek^[11] in their clinical review titled, “Should Airway Pressure Release Ventilation be the primary mode in ARDS?” Chatburn *et al.*^[10] contended that APRV causes “overdistension of the alveoli” (volutrauma) and “repetitive cyclic recruitment.” Chatburn *et al.*’s^[10] arguments of “overdistension” and “repetitive cyclic recruitment” were valid in theory. The findings were based on a “physical breathing simulator” and mathematical models disregarding the fact that different patients respond differently to MV. Similarly, Sasidhar and Chatburn^[12] in a case report discussed their negative experience with APRV. Although the case report was well-written, the clinical and technical approaches used for the initial settings and the methods used for monitoring the VT were flawed. The reason for the large VT variation in Sasidhar and Chatburn^[12] case report is because of the absence of sedation in the initial stage of APRV [Appendix]. The clinical strategy of using sedation and paralytic agents is not new as other clinical studies have suggested the use of sedation and paralytics in the initial stage of managing ARDS patients.^[13,14]

Although spontaneous respirations are essential for the proper use of the APRV at the management and weaning stages, sedation is necessary during the initial phase of APRV to prevent VT fluctuations [Appendix C]. Second, the case report is not enough to justify causality (cause and effect) because more studies are necessary to demonstrate APRV’s safety measures, as Sasidhar and Chatburn^[12] rightfully suggested.

The excessive VT swing may be due to infrequent calibration of the flow sensor by the operator to set up the correct VT, similar to this researcher’s experience with APRV. In addition, the presence of retained secretions creates a turbulent flow that may cause variations in VT. Therefore, respiratory therapy clinicians must understand how to analyze waveforms during MV and apply necessary interventions to minimize VT swings during the application of APRV.

The method whereby the operator sets and checks the automatic tube compensation (ATC) is important in the initial stage of APRV. ATC is an adjunct feature in most ventilator modes to overcome the resistance (R_{aw}) of breathing through an artificial airway.^[15] A pressure gradient is generated

when a patient breathes spontaneously through both ends of endotracheal tubes (ETTs) or any other artificial airway. The pressure gradient must overcome the R_{aw} of breathing through an ETT. ATC makes the mechanism of breathing through an artificial airway easier for mechanically ventilated patients; however, ATC excessive settings may be counterproductive during the initiation stage of APRV [Appendix C]. ATC will overcompensate with large VT swings, and the resulting effect is unnecessary VT fluctuations delivered to the patient's airway. Clinicians should evaluate the amount of VT used during the initial stage. This researcher's usual clinical practice is to deactivate ATC when initiating APRV; otherwise, ATC is necessary during the management and weaning stage of APRV when spontaneous respiration is important [Appendix].

Finally, one of the critical parameters to VT delivery is the proper setting of the slope. The slope is the amount of time it takes to deliver the pressures from P-low to P-high. The operators often set the slope inappropriately. If the slope setting is too long, the delivery of flow needed to deliver the pressures from P-low to P-high will be slow. Conversely, if the slope setting is too short, it may lead to uneven distribution of pressures to the lower bases of the lungs where lung recruitment is necessary.

APRV is a pressure-limited ventilation associated with a large pressure gradient (P-high minus P-low). The patient's VT will vary in relation to his or her pulmonary compliance and decreases with the R_{aw} .^[16] Therefore, it is important for the operator to continuously check the VT in order to implement safer clinical strategies.

The initial setting for the P-low of 15 cm H₂O was excessive in a case report by Sasidhar and Chatburn.^[12] A recent survey of clinical practice by Miller *et al.*^[17] obtained 78% approval among clinicians (respondents) that P-low should be set at 0 cm H₂O in the initial stage of APRV [Appendix]. Although survey research is not generalizable, it provides clinicians with a guide to the initial APRV setting.

Li *et al.*^[2] reported a significant improvement in the reduction of the peak inspiratory pressure, oxygenation index (OI), lung dynamic compliance, functional residual capacity, and extravascular lung water in moderate-to-severe ARDS using APRV. This researcher did not quantify FRC; however, there were improvements in the OI, PaO₂/FiO₂ ratio, and a reduction in extravascular lung water when APRV was initiated in a CLL patient [Figure 1 and Table 1].

Facchin and Fan^[1] described APRV and HFOV as unconventional treatment methods available for ARDS patients. The authors argued that the reason there is a reduction in mortality for ARDS patients is because of the ventilatory strategies that limit pressure and volume to the lungs. This strategy will prevent VILI and is known as the ARDSnet protocol. Facchin and Fan^[1] suggested that methods that limit volumes to 6 ml/kg of the IBW are insufficient to recruit the

alveoli due to repetitive "cyclic collapse and reopening of the alveoli." Their findings indicated that HFOV and APRV are the ideal modes of ventilation because they are both based on an open-lung approach.

A recent multicenter, randomized, controlled trial reported that HFOV may not reduce mortality.^[6] In fact, the trial was stopped prematurely on the recommendation of a data monitoring committee because the inhospital mortality was 47% in the HFOV group with moderate-to-severe ARDS. Young *et al.*^[7] reported a similar finding that HFOV had no significant reduction in mortality in ARDS patients who were on MV over a 30-day period. This evidence further illustrates that APRV should be the mode of choice when treating ARDS patients. A recent recommendation by Fan *et al.*^[5] stated, "We recommend that HFOV not be used routinely in patients with moderate or severe ARDS." The statement reaffirmed that HFOV is not the mode of choice for ARDS patients. The statement was also underscored by the argument that alternative intervention such as APRV is gaining momentum from the statement: "we suggest that adult patients with ARDS receive RM," which is the hallmark of APRV.^[5] The hallmark is the sustained maximal inspiration strategy using 80%–90% of T-high in the respiratory cycle, resulting in the recruitment of the alveolar units.

Habashi^[3] was one of the first to highlight the safety and stability that APRV creates for the alveolar units. He contended that the intermittent release phase in APRV is one of the main advantages of the mode because it can prevent overdistension of the alveoli in ARDS patients. The result is the matching of the ventilation/perfusion ratio leading to better oxygenation. The FRC thereby improves the stabilization of the alveolar units due to lung volume recruitment. This is because both T-high, which is 80%–90% of the respiratory cycle, and P-high create a "near-sustained inflation" that works synergistically to improve the FRC.

Interestingly, a brief release time associated with T-low is an added advantage to the lung units because it can effectively result in trap volume (dynamic hyperinflation or auto-PEEP) that can further serve as a recruitment strategy (measured-PEEP) to help recruit collapsed alveoli. When hypoxemia improves, it follows that ventilation will improve, creating a better acid-base status that can aid in the liberation of ARDS patients from the ventilator.

Maxwell *et al.*^[18] randomized traumatically injured patients who needed MV into a respiratory protocol for APRV or low VT ventilation (LOVT). Sixty-three patients enrolled in the study over a 21-month period. Thirty-one patients were classified into APRV, whereas 32 were grouped into LOVT. The study showed that APRV had a similar safety profile compared to LOVT. Second, the sedation protocol in both study groups was similar. Maxwell *et al.*'s^[18] findings further underscore the importance of sedation when managing ARDS patients.

Mireles-Cabodevila and Kacmarek^[11] in a clinical review article explored the advantages and disadvantages of using APRV; they explained the safety, comfort, and process of “ventilator liberation” (weaning from the ventilator). The researchers in this case report concurred with Mireles-Cabodevila and Kacmarek’s^[11] suggestion that a higher mean airway pressure (P_{aw}) is necessary to improve oxygenation. The higher than normal P_{aw} (usually 25–30 cm H₂O) allows for the continuous recruitment of the alveolar units, resulting in better ventilation/perfusion matching. Subsequently, the PaO₂ improves gaseous exchange.

Clinical implications

In this case report, the researchers followed the ARDSnet protocols^[8] without success. The team ventilated the patient at a rate of 4–6 ml/kg when it was discovered that the patient’s PPLT was >30 cm H₂O. Despite initiating the open-lung strategy (VT 4–6 ml/kg, higher RR of 24–32 BPM, and PEEP between 12 and 18 cm H₂O), the saturation did not improve. In fact, RM was used without much success. The initiation of prone positioning allowed for the weaning of FiO₂ and PEEP transiently; however, this improvement did not last as the patient deteriorated with worsening pulmonary infiltrates resulting in severe hypoxemia [Appendix B]. The initiation of APRV led to the discontinuation of NO within a day. The implication for future research is that APRV works to reduce the extravascular lung water as indicated by the CXR, improving the PaO₂/FiO₂ ratio, improving the OI, and allowing the patient to be more rapidly liberated from the ventilator.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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Appendix: Airway pressure release ventilation clinical protocol

Initial stage 1	Management stage 2	Weaning stage 3
<p>A) Before starting APRV:</p> <ol style="list-style-type: none"> 1) Assure patent airway, for example, clear the airway from excessive secretion 2) Obtain CBC (transfuse blood for Hb <8 g/dl) 3) Avoid lung derecruitment once APRV is started, for example, ventilator circuit disconnection, MOV, or MLT should be performed to ensure no cuff leaks, change endotracheal tube if there is evidence of a broken cuff, use sedation, and paralytics if necessary to enhance spontaneous efforts (minimize VT variation using sedation) 4) Insert A-line and obtain baseline ABG 5) Measure the height of the patient and compute IBW; for males, use $IBW=(\text{Height [cm]} - 100) 0.94$; for females, use $IBW=(\text{Height [cm]} - 100) 0.88$ (use IBW to calculate required VT) <p>B) APRV settings:</p> <ol style="list-style-type: none"> 1) Set P-high=CV P_{PLT} to achieve $VT=8 \text{ ml/kg}$ of IBW (set P-high=PC level in PCV) 2) Set P-low=0 cm H₂O 3) Set T-high=3–6 s (if spontaneous RR start 5–6 s and if no spontaneous RR start 3–3.5 s) 4) Set T-low=0.2–0.5 s (assure measured PEEP is 4–6 cm H₂O for a PaO₂/FiO₂ ratio less than 200 mmHg or 7–8 cm H₂O for a P/F ratio <100 mmHg) 5) Recalibrate flow sensor every 30–60 min to ensure VT consistency at 8 ml/kg 6) Deactivate ATC to set up appropriate VT (do not exceed 8 ml/kg), request sedation and paralytics if necessary <p>C) Safety: Set high-pressure alarm to 6 cm H₂O above measured peak inspiratory pressure (do not exceed 10 cm H₂O above set P-high) and set the high VT alarm 1 ml/kg above the set VT</p> <p>D) Obtain another ABG within 20–30 min of initiation of APRV, then continue to stage 2</p>	<p>A) If saturation does not improve within 30 min or SpO₂ is <88%:</p> <ol style="list-style-type: none"> 1) Increase P-high to achieve $VT=8-10 \text{ ml/kg}$ of IBW for better recruitment (consider P-high 35–40 cm H₂O) 2) Recalibrate the flow sensor to ensure VT consistency 3) Increase T-high by 0.5 s to enhance optimal recruitment 4) Ensure patency of the airway, for example, dislodged endotracheal tube, excessive secretion 5) Monitor leaks around endotracheal tube; use MOV or MLT for cuff leaks if necessary 6) Ensure adequate systemic perfusion, for example, CO, BP, and fluids <p>B) Is PaO₂ <55 mmHg?</p> <ol style="list-style-type: none"> 1) Decrease T-low by 0.1–0.2 s 2) Increase P-low to achieve measured PEEP of 7–8 cm H₂O 3) Recalibrate the flow sensor to ensure 8–10 ml/kg of VT 4) Consider increasing FiO₂, prone positioning, and check systemic perfusion to support CVP, BP, and CO 5) Consider stopping paralytics if oxygenation improves 6) Wean P-high to 30–35 cm H₂O after recruitment (for example, PaO₂ <55 mmHg or SpO₂ <88%) <p>C) Obtain ABG and CXR, and assess vital functions: Oxygenation, ventilation, perfusion, and circulation (act accordingly with the team)</p> <p>D) Improve ventilation by:</p> <ol style="list-style-type: none"> 1) Weaning sedation and stopping paralytics 2) Increase P-high (30–35 cm H₂O) 3) Activate ATC and enter the correct tube size 4) Decrease T-high by increments of 0.2–0.8 s if spontaneous breathing is inadequate by increasing the cycling time 5) If ventilation is adequate, see the waveform to eliminate incomplete RR by increasing the T-high for 0.2–0.8 s 	<p>A) Before proceeding to weaning stage, assure the following:</p> <ol style="list-style-type: none"> 1) Decreased pulmonary infiltrates without air bronchograms as chest radiogram 2) Improved acid-base status 3) Improved GCS of 10–11 with patient responding to verbal commands 4) FiO₂=0.30–0.40 5) Ensure good cough and gag reflex <p>B) Start the weaning process:</p> <ol style="list-style-type: none"> 1) If VT is >8 ml/kg, decrease P-high by 2 cm H₂O 2) If VT <8 ml/kg, decrease T-high by 0.5 s to achieve 3.0–3.5 s 3) Decrease sedation or stop sedation if necessary to enhance spontaneous efforts 4) Check for electrolyte imbalance, especially K, Mg, and phosphates (discuss the nutritional status of the patient with the team; consider a high-protein diet before extubation) 5) If patient deteriorates, continue to stage 2 <p>C) Switch to PSV</p> <ol style="list-style-type: none"> 1) Start PSV 10/10 to achieve VT of 8 ml/kg for the first 2 h (increase PS level if necessary to achieve VT of 8 ml/kg) 2) Decrease PSV by 1–2 cm H₂O every 4–6 h ensuring VT of 6–8 ml/kg 3) Decrease PSV further if VT is >8 ml/kg 4) Check ABG within the hour after each change <p>D) Perform leak test (consider dexamethasone if leaks are <40%), NIF, RR/VT <100; discuss extubating the patient with the critical care team</p>

APRV: Airway pressure release ventilation, CBC: Complete blood count, Hb: Hemoglobin, VT: Tidal volume, ABG: Arterial blood gas, IBW: Ideal body weight, CV: Conventional ventilation, P_{PLT} : Plateau pressure, ATC: Automatic tube compensation, CXR: Chest X-ray, BP: Blood pressure, PEEP: Positive end-expiratory pressure, GCS: Glasgow Coma Scale, FiO₂: Fraction of inspired oxygen, PSV: Pressure support ventilation, RR: respiratory rate MOV: Minimal occlusive volume, MLT: Minimal leak technique, PC: Pressure control, PCV: Pressure control ventilation, CVP: Central venous pressure, CO: Cardiac output, PS: Pressure support, NIF: Negative inspiratory force