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CLINICAL BRIEF

Airway Pressure Release Ventilation: An Alternative Ventilation Mode for Pediatric Acute Hypoxemic Respiratory Failure

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Abstract Airway pressure release ventilation (APRV) is a relatively new mode of mechanical ventilation. The use of this model of ventilation in pediatrics has been limited. The authors describe their experience with this mode of ventilation in a series of pediatric hypoxemic respiratory failure patients. Three patients with acute hypoxemic respiratory failure (AHRF) were treated with APRV, when oxygenation did not improve with pressure control ventilation (PCV). The mean age of the patients was 5.8±1.3 months. Fractional oxygen concentration decreased from 0.97±0.02 for PCV to 0.68±0.12 for APRV, peak airway pressure fell from 36.6±11.5 cm H_2O for PCV to 33.3±5.7 cm H_2O for APRV, mean airway pressure increased from 17.9±5.9 cmH_2O for PCV to 27±2.6 cmH_2O for APRV and release tidal volume increased from 8.3±1.5 mL/kg for PCV to 13.2±1.1 mL/kg for APRV after 1 h. APRV may improve oxygenation in pediatric AHRF when conventional mechanical ventilation fails. The APRV modality may provide better oxygenation with lower peak airway pressure.

Keywords Airway pressure release ventilation · Acute hypoxemic respiratory failure · Oxygenation · Pediatrics · Hypoxemia

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Abbreviations

AHRF	Acute hypoxemic respiratory failure
ALI	Acute lung injury
APRV	Airway pressure release ventilation
ARDS	Acute respiratory distress syndrome
CPAP	Continuous pressure airway pressure
EELV	End expiratory lung volume
FiO_2	Fractional oxygen concentration
MAP	Mean airway pressure
PaO_2	Partial arterial oxygen pressure
PCO_2	Partial carbon dioxide pressure
PCV	Pressure control ventilation
PEEP	Positive end-expiratory pressure
P_{high}	High-pressure setting
PICU	Pediatric intensive care unit
PIP	Peak inspiratory pressure
P_{low}	Low-pressure setting
PS	Pressure support
SIMV	Synchronized intermittent ventilation
SpO_2	Oxygen saturation
VT	Tidal volume
VTe	Release tidal volume
T	Time
T_{high}	Set time interval for P_{high}
T_I	Inspiratory time
T_{low}	Set time interval for P_{low}

Introduction

Acute hypoxemic respiratory failure (AHRF) is one of the hallmarks of acute lung injury (ALI) and acute respiratory distress syndrome (ARDS), which are caused by an inflammatory process initiated by any of a number of

potential systemic and/or pulmonary insults. In these critically sick patients, optimizing the management of oxygenation is crucial. Airway pressure release ventilation, (APRV) is a relatively new mode of mechanical ventilation that has been shown to achieve recruitment and improve oxygenation and gas exchange while maintaining acceptable peak airway pressure (PIP) [1–5]. APRV commences at an elevated baseline pressure and follows with a deflation to accomplish tidal ventilation. The modality consists of a high-pressure setting (P_{high}) and a low-pressure setting (P_{low}) [6, 7]. Recruitment and oxygenation occur during P_{high}—which is a high-level CPAP—at a set time interval (T_{high}). Ventilation is augmented by releasing airway pressure to a second CPAP level (P_{low}).

Previous studies and case reports in adult patients suggest that APRV provides a benefit by allowing oxygenation and ventilation to patients with acute lung injury (ALI) and ARDS [1–5]. There is limited data about the benefits of APRV in children [8–10]. In this paper, authors have presented three patients with AHRF whose oxygenation did not improve with conventional ventilation and were treated with APRV.

Case Reports

Three patients who had developed AHRF were treated with APRV. The underlying causes of the respiratory failure were different. The demographic data and AHRF etiologies are shown in Table 1. Before the application of APRV, patients were mechanically ventilated using pressure control ventilation (PCV) mode with ventilatory variables, with sequentially increased PIP and positive end-expiratory pressure (PEEP), in an attempt to achieve maximum oxygenation at a minimum fraction of inspired oxygen (FiO_2). The ventilator settings before conversion to APRV are shown in Table 2. All patients were ventilated using Dräger Evita XL ventilator (Dräger, Lübeck, Germany). Ventilatory variables on APRV were set according to the Habashi et al's clinical guide for pediatric patients [11].

Case 1 An 11-month-old girl with a past medical history of chronic obstructive lung disease, presented with an episode of wheezing and hypercapneic respiratory failure. She had recurrent wheezing attacks since last 6 months. During the hospitalization, she was intubated for hypercapneic respiratory failure and transferred to our pediatric intensive care unit (PICU). She was hemodynamically stable. Initially the patient was supported with PCV. At the 24th h of mechanical ventilation, the patient's oxygenation deteriorated, partial arterial oxygen pressure/fraction of inspired oxygen (PaO_2/FiO_2) ratio was 85 torr, and the chest radiograph showed right patchy infiltrate and left hyperaeration. *Escherichia coli* was grown from blood cultures. The oxygenation was not improved by high PEEP, high FiO_2, long inspiratory time (T_I) ventilatory settings and prone positioning. On the 32th h of mechanical ventilation, further deterioration in the patient's oxygenation warranted a trial of APRV. Although patients with chronic obstructive lung disease and acute asthma exacerbation maybe difficult to ventilate with APRV, severe hypoxemia warranted the attempt. APRV ventilation resulted in a marked improvement in oxygenation. The FiO_2 level decreased gradually. The patient continued to be weaned from ventilatory support and at the fifth day of APRV, pressure targeted synchronized mandatory ventilation (SIMV) and pressure support (PS) were introduced. The hyperaeration on the left lung persisted, so chest computed tomography and bronchoscopy were performed, leading to the diagnosis of Swyer-James-MacLeod syndrome. The patient was extubated on the 13th day of mechanical ventilation.

Table 1 Demographics and AHRF causes

Patient	Diagnosis	Age	Weight (kg)
Patient I	Nosocomial sepsis	11 months	7.4
Patient II	Metabolic acidosis	3.5 months	4.3
Patient III	Septic shock	6 months	5.8

ALL acute lymphoblastic leukemia

Table 2 Conventional ventilation data of the patients pre-enrollment to APRV treatment

Patients	PIP (cmH_2O)	MAP (cmH_2O)	PEEP (cmH_2O)	FiO_2 (%)	VT
Patient I	30	19	13	90	10
Patient II	30	11	15	100	7
Patient III	50	23	12	90	8.0

PIP peak inspiratory pressure; *MAP* mean alveolar pressure; *PEEP* positive end-expiratory pressure; *FiO_2* fractional oxygen concentration; *VT* tidal volume

Case 2 A three and half month old boy was transported to our PICU with acute encephalopathy and the suspicion of inborn error of metabolism. Severe metabolic acidosis was evident on arterial blood gas analysis at admission (pH: 6.97, bicarbonate level: 8 mmol/L, base deficit: 10 mmol/L). He was intubated and put on PCV. At the 24th h of mechanical ventilation, the oxygenation deteriorated and chest radiograph revealed bilateral diffuse ground glass opacities. The PaO_2/FiO_2 was 100 torr. Oxygenation did not improve and SpO_2 was 80%, despite high PEEP, high FiO_2, long T_I ventilatory settings and prone positioning. APRV ventilation was started. Within an hour, marked improvement in oxygenation was noted and SpO_2 increased to 93%. After the 33th h of APRV, ventilation the lungs were well expanded; pressure

targeted SIMV and PS was started. He was eventually extubated on day 22.

Case 3 A 6-month-old girl, was treated for intractable seizures. She had nosocomial sepsis and hemodynamic instability. She was intubated and received aggressive fluid resuscitation and vasoactive medication infusions. Her hemodynamic condition improved, but at the 18th h of PICU admission, she became tachypneic and the SpO_2 decreased to 88%. Chest radiograph showed diffuse bilateral infiltrates. She was ventilated with high FiO_2, high PEEP and long T_I ventilatory settings. SpO_2 increased but the FiO_2 remained close to 1.00. She was started on APRV at the 26th h after admission to PICU. Her oxygenation improved, oxygen requirements were decreased and ventilatory settings were weaned. After 7 days on APRV, she was put back on conventional ventilation and was extubated on day 9.

Mean time on conventional ventilation before conversion to APRV was 33±10.8 h. The mean time spent on APRV was 108±66.8 h. The ventilatory settings and blood gases of the patients pre-enrollment to APRV and after APRV are compared in Table 3. The PIP decreased, the average mean airway pressure (MAP) increased and release V_T increased after the start of APRV. The pH increased and PCO_2 level decreased with APRV (Table 3). FiO_2 and partial carbon dioxide pressure (pCO_2) level progressively decreased throughout the APRV period.

Discussion

Data obtained from present cases support the notion that APRV can improve oxygenation in some children with AHRF as in Krishan case series [9]. Krishan et al. showed that oxygenation index improved in several of their patients managed with APRV but there was no data about lung mechanics. In Shultz et al. study [10], the MAP and VT_e in children were slightly lower in the APRV mode as compared to SIMV. The present findings differ from Shultz et al. study. In the present patients, the MAP increased to 53% and VT_e increased to 59%, and PIP decreased to 9%, after the start of APRV. Thus the use of APRV in the present patients recruited the lungs by increasing MAP; optimized mean airway pressure/lung volume providing a greater surface area for gas exchange. The APRV modality could provide better oxygenation with lower peak airway pressure.

It is important not only to recruit the lung, but also to keep it open. APRV maybe viewed as a nearly continuous recruitment maneuver with P_{high} providing 80% to 95% of the cycle time creating a stabilized "open lung" [5]. The progressive decrease in FiO_2 throughout the APRV period in the present patients, support the hypothesis that APRV stabilized the "open lung".

Also, authors experience, alveolar ventilation was adequate on APRV. PCO_2 levels improved during APRV. Ventilation in APRV occurs during the release phase and during spontaneous breathing at any point in the cycle. APRV achieves equally effective alveolar ventilation with lower minute ventilation compared to PCV, suggesting less dead space ventilation.

Conclusions

APRV may offer potential clinical advantages for the ventilatory management of pediatric patients and maybe considered as an alternative mechanical ventilation mode in the pediatric population. Also, APRV may improve oxygenation in patients when conventional ventilation has failed to improve oxygenation. APRV is a relatively simple modality that can be implemented easily, particularly when high frequency oscillatory ventilation is not accessible. The effect of APRV on the duration of mechanical ventilation,

Table 3 Conventional ventilation versus APRV

	Pre-enrollment to APRV	APRV time (hours)			
		T_1	T_{24}	T_{48}	T_{72}
PIP/Phigh(cmH_2O)	36.6±11.5	33.3±5.7	30.3±5.5	29±1.4	25.5±2.1
MAP (cmH_2O)	17.9±5.9	27±2.64	24.6±4.16	23.5±0.7	21.6±2.3
PEEP/Plow (cmH_2O)	13.3±1.5	0	0	0	0
T_{high} (sec)		3.6±0.5	3.7±0.4	3.8±0.2	3.65±0.21
T_{low} (sec)		0.73±0.2	0.76±0.11	0.9±0	0.75±0.21
V_{Te} (mL/kg)	8.3±1.5	13.2±1.1	12±1.7	11.0±1.41	9.5±6.7
Set FiO_2 (%)	96.6±2.3	68.3±11.5	60.0±5	57.5±3.5	52.5±3.5
		T_4	T_{24}	T_{48}	T_{72}
pH	7.23±9.7	7.32±9.5	7.35±6.42	7.34±4.9	7.39±1.41
pCO_2, mmHg	72.3±21.4	63.4±16.8	61.4±7.85	56.4±6.43	57.0±12.7

PIP peak airway pressure; *Phigh* high pressure setting; *MAP* mean airway pressure; *PEEP* positive end expiratory pressure; *Plow* low pressure setting; T_{high} high pressure setting time interval; T_{low} low pressure setting time interval; V_{Te} expiratory tidal volume; FiO_2 fractional oxygen concentration; *T* time

duration of PICU stay, and mortality remains to be studied in pediatric patients.

Conflict of Interest We declare that we have no conflict of interest.

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