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Clinical research about airway pressure release ventilation for moderate to severe acute respiratory distress syndrome

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Abstract. – OBJECTIVE: To evaluate clinical effects of airway pressure release ventilation (APRV) in patients suffering from moderate to severe acute respiratory distress syndrome (ARDS).e of a patient presented with significant high aminotransferase levels due to the first human R. aeschlimannii infection ever detected in Italy. The hypothesis of rickettsiosis was made on the basis of a comprehensive medical history and was confirmed by serological tests. Molecular analyses made on a sample of hepatic tissue revealed the presence of a rickettsial species never found before in human liver.

PATIENTS AND METHODS: From August 2012 to August 2014, fifty-two cases with moderate to severe ARDS were randomly divided into two groups. In the first group (APRV) the airway pressure release ventilation was used; the second group (SIMV) was ventilated using synchronized intermittent mandatory ventilation mode and positive end expiratory pressure (PEEP). Changes in oxygenation index, respiratory mechanics, extravascular lung water, functional residual capacity change and hemodynamics were recorded in both groups after mechanical ventilation. TNF-α and IL-10 levels in alveolar lavage were also measured. Acute physiology and chronic health evaluation (APACHE) II and Murray scores were evaluated. Pneumothorax and mediastinal emphysema during ventilation were also recorded. The probability of survival, the duration of ICU stay, days without organ failure and days without sedation were compared.

RESULTS: Conditions in APRV were improved significantly. Oxygenation index was increased, airway peak pressure (Ppeak) was reduced, the lung dynamic compliance improved, extravascular lung water was relieved, functional residual capacity increased and Murray score was improved. In APRV group ventilation central venous pressure (CVP) and systemic circulation resistance index (SVRI) were reduced, but cardiac index (CI) increased, and at the same time lac and oxygen saturation of central venous blood ($ScvO_2$) were improved. Free sedatives days were significantly reduced in APRV group while days without mechanical ventilation were increased and days in ICU were shortened significantly. TNF-α and IL-10 concentrations in the alveolar lavage, probability of survival and days without organ failure were similar in both groups.

CONCLUSIONS: In patients suffering from moderate to severe ARDS, application of APRV improved lung function and hemodynamics. It also reduced the need for sedatives and the duration of mechanical ventilation as well as days in ICU.

Key Words

Airway pressure release ventilation, Acute respiratory distress syndrome, Adult, Free of mechanical ventilation within 28d, Mortality.

Abbreviations

APRV: airway pressure release ventilation; SIMV: synchronized intermittent mandatory ventilation; APACHE: Acute Physiology and Chronic Health Evaluation; HR: heart rate; MAP: mean arterial pressure; CVP: central venous pressure; ELVI: extravascular lung water index; $PA\text{-}DO_2$: Difference of Alveoli-arterial oxygen pressure; R: Resistance; Cdyn: dynamic compliance; Ppeak: airway peak pressure; Pmean: airway mean pressure; FRC: functional residual capacity; CI: Cardiac index; SVRI: systemic circulation resistance index; $ScvO_2$: Oxygen Saturation of central venous blood; Lac: Lactate.

Introduction

In recent years, great progress has been made in the mechanical ventilation techniques for acute respiratory distress syndrome (ARDS); however, the mortality rate of ARDS is still high (40%).

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Currently, lung protective ventilations, including open lung and low tidal volume, are among major ARDS mechanical ventilation strategies. In-depth studies showed that the prognosis of ARDS is closely related to mechanical ventilation airway pressure. Even low tidal volume used for severe ARDS treatment can lead to a high airway pressure, resulting in poor prognosis. Meanwhile, open lung and low tidal volume lung protective ventilations for ARDS cannot improve oxygenation and may require higher levels of sedatives and analgesic agents, which may result in hypercapnia[1]. Airway pressure release ventilation (APRV) can be used for alveolar recruitment and maintaining by setting high pressure (Phigh) and make alveolar with slow time constants remain open by low pressure for (Plow) hence eliminating CO_2. In theory, APRV can be effective in re-expansion of collapsed lung tissue and it can maintain the maximum and persistent alveolar re-expansion. In patients with ARDS, the APRV is proved to be effective in improving the respiratory function without causing any side-effects on the circulatory function, and this is highly beneficial to ARDS patients[2-4]. In the present study, we compared APRV to small tidal volume protective ventilation strategy for providing a reliable basis for the clinical application of APRV by observing respiratory and circulatory function changes. We monitored pulmonary inflammatory changes under the two methods of mechanical ventilation while observing and monitoring the clinical prognostic indicators.

Patients and Methods

Patient preparation

Endotracheal intubation or tracheotomy was conducted for mechanical ventilation. All patients received mechanical ventilation therapy with AVEA ventilator. Patients were in the synchronized intermittent mandatory ventilation mode and positive end expiratory pressure (SIMV+PEEP) ventilation mode. Mechanical ventilation parameters were adjusted on the basis of the analysis of arterial blood gas and pulse oxygen saturation (SpO_2) with tidal volume 6 ml to 8 ml per kg, plateau airway pressure under 35 cmH_2O, respiratory frequency of 14 to 20 times per minute, minute ventilation volume equal to 4L to 10L per min and regulating FiO_2 between 40% to 100%. The ventilator setting parameters and airway peak pressure (Ppeak) were recorded to determine the low turning point by the low flow rate method recording the static PV curve. A deep venous catheter (PULSION Medical Systems SE, Feldkirchen, Germany) was attached to PiCCO machine (PULSION Medical Systems SE, Feldkirchen, Germany) for blood gas analysis, including central venous pressure, cardiac output and extravascular lung water index (ELWI). During the observation period, patients were given appropriate sedation and analgesia. Sedation RASS score was -2 to +1.

Random grouping

Patients were randomly divided into two groups according to random number table. Twenty-six patients enrolled in APRV group underwent the airway pressure release ventilation and twenty-six patients in SIMV group received the small tidal volume lung protective ventilation. We set PEEP according to the quasi-static PV curve determination of lower inflection point (Pflex). Patients in both groups were treated with sustained inflation before ventilation. The ventilator mode and parameters were set according to the grouping after recruitment. PEEP was P_{flex}+2 cmH_2O.

Low velocity method tracings quasi-static PV curve

Full sedation or muscle relaxants were given when necessary. Respiratory frequency was 4 to 5 times per minute, and tidal volume was 15 ml per kg in volume control ventilation. The inspiratory flow was adjusted to 5 L per minute. We recorded the PV curves according to the ventilator monitor screen. We performed automatic measure lower inflection point for three times and we recorded the average value.

Sustained inflation

FiO_2 was adjusted to 100% before recruitment. Continuous airway positive pressure (CPAP) was conducted after the airway secretions were fully cleared. Airway pressure of 30 cm H_2O was maintained for 20 seconds.

APRV settings

Airway high mean pressure (P high) was preset to 30 cmH_2O. Time of high duration (T high) was set to 4 to 8 seconds. Airway low mean pressure (Plow) was 0 cmH_2O, Time of low duration (T_{low}) was set to 0.4 to 0.8 second. PEEP was set to P_{flex} +2 cmH_2O. When the high airway pressure was less than 16 cmH_2O, and the total time of APRV high pressure was gradually shortened until it was converted to CPAP.

Measurement of increased in functional residual capacity

Increased lung volume were measured 12h, 24h, 48h and 72h after mechanical ventilation. Pressure-volume (P-V) curve method was used for measuring the increased lung volume[5]. Respiratory frequency was reduced to at least 6 times per min. Expiratory time was prolonged to 9 seconds. PEEP was adjusted to 0 cmH_2O at the end of inspiration to determine the tidal volume. The difference was equal to the increased functional residual capacity (ΔFRC).

Observation index

Respiratory mechanics, oxygen synthetic index and ELWI: Respiratory mechanics of the two groups were monitored through the ventilators at the time of mechanical ventilation, also 12h, 24h, 48h and 72h after mechanical ventilation. Functional residual capacity and the dynamic compliance were measured at the same. We monitored arterial blood gas analysis and recorded the difference of Alveoli-arterial oxygen pressure ($P_{A\text{-}D}O_{2)}$ and calculated the oxygenation index[2]. We also monitored ELWI through PiCCO during mechanical ventilation as well as 12h, 24h, 48h and 72h after mechanical ventilation. CVP and lactate were also monitored. 3. Pulmonary inflammatory reaction: Fiber bronchoscopy was used 24h, 48h and 72h after ventilation in both groups and 5 ml of normal saline were added to the inferior lobe lung. Lavage fluids were examined using ELISA to obtain a reading on inflammatory factors TNF- α and IL-10 concentrations[4]. We also monitored the APACHE II and Murray scores of acute lung injury at the time of mechanical ventilation and repeated monitoring in 1, 2, and 3 days after ventilation. We recorded ventilator-induced lung injury including pneumothorax, mediastinal emphysema and other serious incidents during ventilation[5]. We as well registered 28-days survival rate, 28-days off-ventilator time, time without organ failure and sedation time.

Statistical Analysis

All data were analyzed by SPSS 12.0 statistical software (SPSS Inc., Chicago, IL, USA). Measurement data were presented by $x\pm s$. A *t*-test was applied for comparisons between groups. One-way analysis of variance (one-way ANOVA) was used for comparison between each group. Pearson X^2-test was applied in comparisons between groups. 28 days survival rate was measured using Kaplan-Meier survival analysis. $p<0.05$ was considered to be statistically significant.

Results

General situations

Differences between the two groups on age, gender, disease composition, oxygenation index and lactate, hemodynamics, APACHE II score, and Murray score were not statistically significant ($p>0.05$) (Table I).

Table I. Comparison of general data in APRV and SIMV groups.

		APRV Group (n=26)	SIMV Group (n=26)	Statistical value	*p*-value
Age		54.3±8.4	53.6±9.5	t=0.290	0.821
Pathology	Lungs	8	8	×2=0.168	0.723
	Trauma	4	4		
	Post-oper.	4	4		
	Shock	7	7		
	Pancreatitis	1	2		
	Other	2	1		
APACHE		18.5±4.6	17.7±6.7	t=0.128	0.876
Murray		3.3±0.5	3.2±0.5	t=0.811	0.442
HR (time.min^{-1})		98±15	101±21	t=1.270	0.301
MAP (mmHg)		84±13	83±16	t=0.501	0.543
PaO_2/FiO_2 (mmHg)		119±35	118±36	t=0.651	0.468
CVP (mmHg)		8.7±3.1	9.1±2.8	t=0.507	0.556
Lac ($mmoll^{-1}$)		3.2±0.8	3.1±1.1	t=0.143	0.823
ELVI (mlkg)		19.5±4.6	19.7±5.5	t=0.146	0.812

Note: compared with Group SIMV, *p <0.05

Table II. Comparison of respiratory mechanics, oxygenation index and ELWI.

Index	Group	0	12h	24h	48h	72h
Ppeak (cmH_2O)	APRV	30.7±5.4	25.9±5.8*	25.3±5.1*	23.2±4.6*	23.7±5.2
	SIMV	29.5±6.9	29.2±5.3	28.5±6.2	26.1±5.6	24.5±6.2
	t	0.698	2.142	2.032	2.040	0.504
	p	0.567	0.035	0.047	0.047	0.768
Pmean	APRV	22.5±3.3	22.3±4.6*	21.2±5.3*	19.4±4.6*	18.3±3.5
	SIMV	22.7±3.6	20±3.4	18.2±5.1	17.1±3.3	17.2±2.6
	t	0.209	2.050	2.080	2.072	1.286
	p	0.875	0.047	0.046	0.046	0.324
C_{dyn} ($ml.cmH_2O^{-1}$)	APRV	32.5±7.6	42.7±6.7*	45.5±4.6*	51.7±5.7*	51.5±4.6
	SIMV	31.7±7.5	37.8±6.5	42.7±3.8	47.6±8.5	49.7±5.5
	t	0.382	2.677	2.393	2.043	1.280
	p	0.786	0.011	0.019	0.042	0.326
R ($cmH_2O.l^{-1}.s^{-1}$)	APRV	13.5±2.6	10.1±3.5*	9.5±2.3*	8.7±2.6*	8.3±3.5
	SIMV	13.7±3.1	12.2±3.5	11.7±2.6	10.6±2.3	9.2±2.6
	t	0.252	2.163	3.231	2.791	1.053
	p	0.871	0.038	0.015	0.038	0.354
PaO_2/FiO_2 (mmHg)	APRV	119±35	158±47*	178±46*	213±48*	220±46
	SIMV	118±36	135±34	153±38	183±55	212±55
	t	0.102	2.022	2.136	2.095	0.569
	p	0.923	0.047	0.043	0.045	0.518
$P_{A-D}O_2$ (mmHg)	APRV	265±53	191±57*	176±46*	155±52*	152±46
	SIMV	249±46	226±55	207±63	189±68	163±50
	t	1.163	2.253	2.026	2.025	0.826
	p	0.189	0.031	0.047	0.047	0.152
ELWI ($mlkg^{-1}$)	APRV	19.5±4.6	16.3±4.7*	15.1±4.6*	12.7±3.7*	12.5±3.6
	SIMV	19.7±5.5	18.8±3.5	17.4±3.3	14.8±3.5	12.7±3.3
	t	0.142	2.175	2.072	2.102	0.104
	p	0.827	0.032	0.046	0.044	0.921

Note: compared with Group SIMV, *p<0.05

Respiratory mechanics, oxygenation index, and ELWI

PaO_2/FiO_2 in APRV group before and 48h after ventilation increased while Ppeak decreased. The static adaptation and functional residual capacity were improved (p<0.05). The indexes by the time of 72h showed no significant differences. Pmean in the APRV group increased more than that of Group SIMV (Table II). At the same time, ΔFRC was significantly improved in APRV group (Table III).

Hemodynamic changes

SVRI and CVP in APRV group decreased, while CI increased significantly (p<0.05). Lac and $ScvO_2$ were improved significantly 48h before ventilation (p<0.05). MAP and HR showed no significant differences (p>0.05) (Table IV).

Lung inflammatory reaction

The comparison on TNF-α and IL-10 concentrations in two groups did not reveal any significant differences (p>0.05) (Table V).

APACHE II score and Murray acute lung injury score

Murray acute lung injury score showed improvement 1 day and 2 days after ventilation (p>0.05) (Table VI).

Comparison of 28d off-ventilator time, time in ICU, time with no organ failure and time without sedation

No pneumothorax, mediastinal emphysema and other severe ventilator-induced lung injuries were observed during mechanical ventilation.

Table III. Comparison of ΔFRC (ml) changes.

Group	12h	24h	48h	72h
APRV	75±36*	117±47*	135±46*	147±37
SIMV	47±35	78±45	92±38	138±35
t	2.843	3.056	3.589	0.901
p	0.015	0.032	0.001	0.421

Note: compared with Group SIMV, *p<0.05

Table IV. Comparison of hemodynamic indexes.

Index	Group	0	12h	24h	48h	72h
HR (time.min^{-1})	APRV	98±15	95±21	90±17	88±15	90±21
	SIMV	101±21	93±26	94±19	94±18	95±26
	t	0.593	0.305	0.800	1.305	0.763
	p	0.708	0.654	0.548	0.198	0.452
MAP (mmHg)	APRV	84±13	82±14	90±17	88±16	87±15
	SIMV	83±16	81±13	88±16	87±15	82±16
	t	0.247	0.267	0.437	0.232	1.162
	p	0.683	0.702	0.558	0.692	0.323
CVP (mmHg)	APRV	8.7±3.1	7.6±1.7*	7.5±1.6*	6.7±1.7*	6.5±1.6
	SIMV	9.1±2.8	8.8±2.5	8.7±1.8	8.0±1.5	6.7±1.5
	t	0.488	2.024	2.541	2.924	0.465
	p	0.596	0.047	0.017	0.009	0.589
CI	APRV	2.7±0.6	3.2±0.5*	3.3±0.4*	3.6±0.5*	3.7±0.4
(l.min^{-1}·m^{-2})	SIMV	2.8±0.7	2.9±0.5	3.0±0.6	3.1±0.4	3.5±0.7
	t	0.553	2.163	2.121	3.982	1.264
	p	0.601	0.043	0.043	0.001	0.196
SVRI	APRV	1695±274	1356±323*	1348±314*	1390±317*	1334±328
(dyn.s.m^2.cm^{-5})	SIMV	1685±267	1634±321	1628±343	1590±297	1484±313
	t	0.133	3.113	3.070	2.348	1.692
	p	0.897	0.003	0.004	0.043	0.102
Lac	APRV	3.2±0.8	2.3±0.5*	2.0±0.6*	1.1±0.4	1.2±0.5
(mmoll^{-1})	SIMV	3.1±1.1	2.8±0.4	2.5±0.6	1.2±0.3	1.1±0.4
	t	0.375	3.982	3.004	1.019	0.796
	p	0.677	0.001	0.031	0.158	0.414
$ScvO_2$	APRV	58±14	62±7*	67±6*	68±5	68±4
	SIMV	52±14	57±9	62±8	67±6	67±5
	t	1.545	2.236	2.550	0.653	0.796
	p	0.167	0.034	0.017	0.509	0.478

Note: compared with Group SIMV, *$p<0.05$

28d off-ventilator time in APRV group increased more prominently (19.7+8.3 days) compared to the other group (16.1+ 1 days). The time in ICU was also shortened in APRV group (7.5+3.5 days) when compared to the second group (9.5 + 3.2) days ($p<0.05$). There were no significant differences between the two groups in the case of time of no organ failure ($p>0.05$) (Table VII).

Comparison of 28 days survival rate

Eight patients (28.5%) in the group APRV died while nine cases (34.6%) in the group SIMV lost their lives within 28 days. The differences were not statistically significant ($p>0.05$).

Discussion

In theory, APRV can improve the respiratory function of ARDS and reduce Ventilator-induced Lung Injury (VILI). Presently, there are several existing studies about mechanical ventilation strategies in ARDS. These studies explained a few strategies that were designed to: i) promote full recruitment of collapsed alveolar caused by ARDS, ii) to improve oxygenation, and iii) preventing VILI due to improper mechanical ventilation as the basic points of mechanical ventilation. APRV is a type of improved continuous airway positive pressure (CPAP) system, which is a pressure-control, time-trigger, pressure-limited and time-switch type of ventilation mode by adding a pressure release valve at the expiratory end. It allows patients spontaneously breathing during the entire respiratory cycle[6]. APRV improves oxygenation by the means of increasing: i) based CPAP pressure levels, ii) the duration of hypertension, and iii) oxygen concentration (FiO_2). CO_2 discharge acts via increasing the APRV release rate and pressure. The way APRV influenced the end expiratory lung volume (EELV) is different from other conventional ventilations. Conventionally, inspiratory

Table V. Comparison of Lung inflammatory reaction indexes.

Index	Group	0	12h	24h	48h	72h
HR	APRV	98±15	95±21	90±17	88±15	90±21
(time.min^{-1})	SIMV	101±21	93±26	94±19	94±18	95±26
	t	0.593	0.305	0.800	1.305	0.763
	p	0.708	0.654	0.548	0.198	0.452
MAP	APRV	84±13	82±14	90±17	88±16	87±15
(mmHg)	SIMV	83±16	81±13	88±16	87±15	82±16
	t	0.247	0.267	0.437	0.232	1.162
	p	0.683	0.702	0.558	0.692	0.323
CVP	APRV	8.7±3.1	7.6±1.7*	7.5±1.6*	6.7±1.7*	6.5±1.6
(mmHg)	SIMV	9.1±2.8	8.8±2.5	8.7±1.8	8.0±1.5	6.7±1.5
	t	0.488	2.024	2.541	2.924	0.465
	p	0.596	0.047	0.017	0.009	0.589
CI	APRV	2.7±0.6	3.2±0.5*	3.3±0.4*	3.6±0.5*	3.7±0.4
(l.min^{-1}·m^{-2})	SIMV	2.8±0.7	2.9±0.5	3.0±0.6	3.1±0.4	3.5±0.7
	t	0.553	2.163	2.121	3.982	1.264
	p	0.601	0.043	0.043	0.001	0.196
SVRI	APRV	1695±274	1356±323*	1348±314*	1390±317*	1334±328
(dyn.s.m^2.cm^{-5})	SIMV	1685±267	1634±321	1628±343	1590±297	1484±313
	t	0.133	3.113	3.070	2.348	1.692
	p	0.897	0.003	0.004	0.043	0.102
Lac	APRV	3.2±0.8	2.3±0.5*	2.0±0.6*	1.1±0.4	1.2±0.5
(mmoll^{-1})	SIMV	3.1±1.1	2.8±0.4	2.5±0.6	1.2±0.3	1.1±0.4
	t	0.375	3.982	3.004	1.019	0.796
	p	0.677	0.001	0.031	0.158	0.414
$ScvO_2$	APRV	58±14	62±7*	67±6*	68±5	68±4
	SIMV	52±14	57±9	62±8	67±6	67±5
	t	1.545	2.236	2.550	0.653	0.796
	p	0.167	0.034	0.017	0.509	0.478

Note: compared with Group SIMV, *p<0.05

increases lung capacity to discharge CO_2. However, APRV achieves this purpose by reducing the end expiratory lung volume, which can avoid lung volume injury caused by a larger static lung capacity. Phigh mode of APRV is a high pressure that is the basis of the CPAP, which is conducive to the collapsed alveoli. Although the setting pressure is high, the airway pressure is low compared to other modes due to the presence of alveoli recruitment so as to reduce lung barotraumas risk. Plow mode is in relatively lower pressure, which is the sustained pressure after the release of high pressure. It can keep CPAP at a level to be helpful for maintaining alveoli open, and to reduce shear injuries[7-9].

Table VI. Comparison of APACHE II score and Murray acute lung injury score.

Index	Group	0	24h	48h	72h
APACHE	APRV	18.5±4.6	17.3±5.8	16.3±6.1	13.9±3.2
	SIMV	17.7±6.7	17.4±5.4	16.5±6.0	14.3±4.2
	t	0.502	0.064	0.120	0.386
	p	0.672	0.934	0.899	0.734
Murray	APRV	3.3±0.5	2.6±0.4*	2.1±0.6*	1.2±0.5
	SIMV	3.2±0.5	2.9±0.4	2.5±0.6	1.1±0.4
	t	0.721	2.704	2.404	0.796
	p	0.453	0.003	0.016	0.432

Note: compared with Group SIMV, *p<0.05

Table VII. Comparison of 28-days off ventilator time, time in ICU, time with no organ failure and time with no sedative.

Group	Time with no sedative (d)	off ventilator time(d)	time in ICU (d)	time with no organ failure (d)
APRV	22.4±8.5*	19.6±8.2*	7.4±3.3*	15.2±3.3
SIMV	17.1±8.6	15.1±8.9	9.5±3.2	14.5±4.2
t	2.221	2.021	2.223	0.800
p	0.036	0.046	0.035	0.548

Note: compared with Group SIMV, *$p<0.05$

At the same time, APRV does not adversely affect the hemodynamics and may even improve them. It is thought that, compared with conventional ventilation, increased intrathoracic pressure may inhibit venous from returning to the heart which leads to reduced cardiac output due to the higher mean airway pressure of APRV. Kamath et al[10] studied HR, CVP, BP, IVF and urine volume of ALI children under APRV mode. Their results showed that measured values were not different compared to those in the traditional mode. By reviewing the literature concerning the APRV in the past 10 years, Calzia and Pradermacher[11] found no report on the existence of any serious damages to the hemodynamics using APRV. Likewise, the presence of spontaneous breathing not only reduces the sedatives usage, but also decreases airway pressures and increase the heart and intrathoracic great vessels' pressure. So it will be conducive to the venous return and increased CI, which improves oxygen delivery and organ perfusion[12,13].

Our results showed that APRV helped ARDS patients with increased PaO_2/FiO_2 of 48 hours. At the same time, Ppeak was reduced, static adaptation was improved and functional residual capacity was enhanced. No significant differences were observed for 72h indexes. Therefore, our results showed that APRV, compared to the SIMV mode, could better improve the early lung function. APRV increased lung volume and improved oxygenation and lung compliance. In theory, APRV can also reduce VILI. The bronchoalveolar lavage inflammatory factor in APRV group had no significant differences compared to the SIMV mode of small tidal volume lung protection combined with pulmonary re-expansion ventilation.

In the APRV group, P_{mean} was higher but the CVP was lower, which may be associated with increased lung volume. At the same time, in the APRV group the higher CI and lower SVRI increased the blood flow perfusion in the tissue. This study also showed that $ScvO_2$ was higher in APRV group and lac was lower. Prior studies showed that application of APRV could increase the cardiac output and blood pressure, which may further help our conclusion that the APRV can be considered as a treatment method for patients with hemodynamics damages. Experiments on animal demonstrated that APRV caused no damage to the hemodynamics and tissue oxygenation, which can be considered a confirmation of our conclusions[12,13].

One may speculate that the main reason that APRV can reduce the duration of mechanical ventilation and shorten the ICU stay is because APRV improves respiratory function at early stages and improve organ perfusion. Thus, spontaneous breathing is preserved with decreasing the sedative use time, which promotes early recovery[14,15].

Conclusions

In summary, APRV, as a mechanical ventilation treatment for moderate or severe ARDS patients, provided better and early respiratory support while improving the lung function. Additionally, APRV: i) improved hemodynamics and tissue perfusion, ii) decreased the sedative use time, iii) reduced the duration of mechanical ventilation, and iv) shortened the duration of ICU stay. Nevertheless, our sample size was small, and studies with larger sample sizes can help us to better explore the possibilities offered by airway pressure release ventilation.

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Conflict of Interests:
The Authors declare that they have no conflict of interests.

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