Original Article

Airway Pressure Release Ventilation in Acute Respiratory Distress Syndrome

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Abstract

Background : *Acute respiratory distress syndrome (ARDS) is characterized by a heterogeneous pattern of diffuse alveolar damage resulting from either a direct, indirect, or a combination of insults to the lungs. The injured lung in acute respiratory distress syndrome (ARDS) is heterogeneous in gas and fluid distribution, depending upon the causes of ARDS.*

Objectives : *To observe the effectiveness of airway pressure release ventilation in acute respiratory distress syndrome.*

Materials & Methods: *This was a prospective randomized controlled trial, was carried out in the Department of Anesthesiology, Analgesia, Palliative and Intensive Care Medicine, Dhaka Medical College Hospital, Dhaka, Bangladesh carried out from the period January 2015 to December 2016, conducted with mechanically ventilated patients with acute respiratory distress syndrome at intensive care unit, DMCH.*

Results: *A total of 48 patients were enrolled in this study and they were divided into two groups. Each group had 24 patients. PaO2, SpO2, mean airway pressure and PaO2/FiO2 were significantly increased (improved) in both groups but significantly higher in APRV group. PaCO2, FiO2 and peak inspiratory pressure were significantly decreased in both groups and significantly lesser in APRV group. Less sedation was required in APRV group.*

Conclusion: *Airway pressure release ventilation is a better and acceptable method of ventilation in acute respiratory distress syndrome.*

Keywords: *Airway Pressure, Release, Ventilation, Acute Respiratory Distress Syndrome*

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Introduction

Acute respiratory distress syndrome (ARDS) is distinguished by dispersed alveolar damage resulting from insults to the lungs[1]. Well described by Gattinoni and colleagues, the injured lung in acute respiratory distress syndrome (ARDS) is heterogeneous in gas and fluid distribution, with variable computed tomography findings within individual patients, depending upon the causes of ARDS[2,3]. With direct insults (e.g. pneumonia, aspiration of gastric content) multifocal parenchymal involvement is found, whereas indirect insults (e.g. sepsis, septic shock, transfusion related

lung injury etc.) result is a more diffuse, interstitial pattern of injury[4].The first case of ARDS was reported in 1967 [5] and included 12 patients with refractory hypoxemia and diffuse infiltrates on chest x-ray. Seven patients died and autopsy findings revealed dense infiltration of the lungs with an inflammatory exudate. There was no evidence of infection, which indicated that ARDS was an acute inflammatory lung injury. In the early stages of acute respiratory distress syndrome (ARDS), multiple areas of the lungs collapse, most often in the dependent regions. There is loss of functional surfactant, creating a condition in which alveolar units are unstable and prone to unopposed surface tension. It results in a decreased volume of aerated lung, intrapulmonary shunting, and, therefore, poor oxygenation. Three phases of ARDS has been recognized, exudative phase, proliferative stage, and fibrotic stage respectively. First phase (exudative phase) develops during the first week. It is distinguished by protein rich and often hemorrhagic, interstitial as well as alveolar edema and hyaline membrane formation. There is necrosis of alveolar type l and endothelial cells. Proliferative phase is characterized by organized of exudates, more deranged architecture, obliterative end arteritis, alveolar type-II cell proliferation, and fibroblasts seen the interstitial as well as in the alveolar space resulting in narrowing and even obliteration of air space. This phase is seen in the second and third week of disease process. Three weeks later fibrotic phase appears after the onset the symptoms but the process can commence promptly from ten days after initiating injury. Gattinoni et al. 2006 [6] showed that the percentage of recruitable lung could range from a negligible fraction to 50 % or more. The choice of recruitment maneuver is based on the individual patient and the ventilatory mode[7]. High tidal volume ventilation may cause hyperinflation of relatively normal regions of aerated lung. Since nonaerated lung tissue is stiffer than normal lung tissue, compliance is reduced and airway pressure is increased. Lung over distension causes direct physical damage, with disruption of the alveolar epithelium and capillary endothelium as well as the induction of an inflammatory response, with the release of cytokines and another mediators[8-10]. Some evidence suggests that the inflammatory response induced in ventilator-induced lung injury has systemic consequences, contributing to the pathogenesis of multisystem organ failure in patients with ARDS. In the treatment of ARDS, different ventilatory modes e.g. lung protective ventilation, pressure-controlled ventilation, airway pressure release ventilation, high frequency oscillatory ventilation can be applied. Advantages of lung protective ventilation includes to limit volutrauma, biotrauma and atelectrauma. The major disadvantage of pressure-controlled ventilation is the decrease in alveolar volume that occurs when there is an increase in airway resistance or a decrease in lung compliance. High frequency oscillatory ventilation delivers small tidal volume (1-2ml/kg) using rapid pressure oscillations (300 cycle /min). High frequency oscillatory ventilation requires a specialized ventilator, and is not available in all hospitals[11]. Airway pressure release ventilation (APRV), a mode of mechanical ventilation that can be useful in situations in which, due to ARDS, the lungs need to be recruited and held open. Airway pressure release ventilation was described by Stock and Downs in 1987[12, 13] as a continuous positive airway pressure (CPAP) with an intermittent release phase. The release phase results in alveolar ventilation and removal of carbon dioxide. Airway pressure release ventilation (APRV) facilitates both oxygenation and carbon dioxide clearance and originally was described as an improved method of ventilatory support in the presence of acute lung injury and inadequate carbon dioxide ventilation[12,13]. In this pressure-limited, time-cycled mode of ventilation, alveolar recruitment takes place over extended periods of inspiration. Effect of spontaneous ventilation, boost up patient's tolerance, support in recruitment in dependent lung areas, greater venous return, soaring

glomerular filtration rate (GFR), recovered small-bowel perfusion and decreased sedation requirements. High mean pressure causes lung recruitment, leading to greater oxygenation, reduction to left ventricular transmural pressure and therefore reduction of left ventricular after load. New generations of ventilators capable of using APRV make this modality readily available in the critical care setting. ARDS is an important cause of acute respiratory failure that is often associated with multiple organ failure. Management of acute respiratory distress syndrome (ARDS) is a challenging task in ICU. ARDS is associated with high mortality rate. Oxygenation is better with APRV with spontaneous breathing than with mechanical ventilation alone. APRV improves matching of ventilation and perfusion. Airway pressure release ventilation is consistent with lung protection strategies that strive to limit lung injury associated with mechanical ventilation. Patient improvement is better in airway pressure release ventilation. There is less complication and no additional cost is required in this mode. So this study was designed to observe the effectiveness of airway pressure release ventilation in acute respiratory distress syndrome and to test hypothesis was airway pressure release ventilation is more effective method of ventilation in acute respiratory distress syndrome.

Methodology And Materials

This prospective randomized controlled trial, was carried out in the Department of Anesthesiology, Analgesia, Palliative and Intensive Care Medicine, Dhaka Medical College Hospital, Dhaka, Bangladesh from January 2015 to December 2016, was conducted with mechanically ventilated patients with acute respiratory distress syndrome at intensive care unit, DMCH. Sampling was done according to availability of the patients and strictly considering the inclusion and exclusion criteria. Diagnosis of ARDS was made on the basis of clinical history, chest X-ray and PaO_2/FiO_2 ratio. For prevention of biasness ARDS (mild and moderate) patients were selected (who receiving $FiO_2 \geq 0.6$ and positive end expiratory pressure > 10 cm H_2O) got admitted into intensive care unit in DMCH. Exclusion criteria were pregnancy, age ≤ 18 years and ≥ 50 years, presence of bronchopleural fistula, an immune-compromising disorder such as AIDS, cirrhosis of liver, terminal cancer, patients with profound airflow limitation including, restrictive lung disease, emphysema, severe bronchospasm, patients with or suspected of having an untreated pneumothorax, recent lung resection, those with profound haemodynamic instability.

Study Procedure:

On enrollment, patient's age, sex, height, weight was recorded. Berlin criteria was used for diagnosis of ARDS. Patients were divided randomly into two groups for mechanical ventilation. One group for airway pressure release ventilation for APRV (group I) and another group for V-SIMV (group II). Data was taken baseline, 6 hours later, and then daily from day 2 to day 6.

Airway pressure release ventilation (APRV) (group I):

In APRV group, high pressure (P_H) setting was adjusted to equal the peak air way pressure on pressure control ventilation or plateau pressure from volume control ventilation. The low pressure (P_L) setting was set at zero by convention. Time spent at high pressure (T_H) was set 4-6 seconds. Duration of the low pressure (T_L) setting was 0.6-0.8 seconds. Initially FiO_2 was 1 then it was decreased gradually. When PaO_2 < 65 mm Hg and /or arterial oxygen saturation SpO_2 < 92%, high pressure (P_H) was increased by 2 cm of H_2O, followed by an increase in (T_H) by 0.5 seconds and then an increase in FiO_2 by 0.1. This cycle was repeated as necessary to restore arterial oxygen levels. If CO_2 > 50 mm Hg and arterial P^H < 7.35, then high pressure (P_H) was increased and time spent in high pressure (T_H) was subsequently decreased.

V-SIMV (group II): Predicted body weight was calculated and initial tidal volume (V_t) was set at 6ml/kg while on SIMV with pressure support. Initial minute ventilation was set at 6 L and the ventilator rate was determined by dividing this amount by the set tidal volume. PEEP and PSV were set at 10 cm of H_2O. 100% O_2 was set initially and gradually it was reduced until FiO_2 0.5. If spontaneous respiratory were >26 breath per minute, the ventilator rate, and/or pressure support were adjusted. For hypoxic conditions, PEEP was increased in 2 cm H_2O increments, repeated twice as necessary, followed by an increase in FiO_2 of 0.1. This cycle was repeated as necessary until $PaO_2 \geq 65$mm Hg or $SaO_2 \geq$ 92%. Respiratory acidosis was treated by increasing the ventilator rate by two breaths per minute.

FiO_2: It was recorded at the time of allocation of APRV and SIMV and it was recorded after 6 hours later and recorded daily for 6 days. If there is develop acid- base disorder, then it was recorded 2 hourly.100% O_2 as inspired gas may lead to absorption atelectasis in lung in distal to the site of airway closure. Absorption atelectasis may occur in as short a time as 6 minutes with 100% O_2 and 60 minutes with 85 % O_2. We tried to keep FiO_2 less than 0.5 to avoid oxygen toxicity without features of hypoxemia. SpO_2 was measured 2 hourly and kept it > 92%.

Measuring of ABG value and ventilator data: ABG values and ventilator measurements (mean airway pressure, peak inspiratory pressure) were recorded and compared baseline parameter, 6 hours later, then for the first 6 days of ventilation. From ABG, we got data on pH, PaO_2, $PaCO_2$, HCO_3^-. Value of PaO_2, $PaCO_2$, HCO_3^- were kept in normal physiological limit. When $ETCO_2 \geq 50$ mm Hg then ABG was done 2 hourly until $PaCO_2$ within normal range. Just after ABG, PaO_2/FiO_2 was calculated.

Sedation: Patients were sedated with an intravenous infusion of fentanyl and supplemented with midazolam infusion according to a preexisting ICU protocol. Sedation levels was maintained by the Motor Activity Assessment Scale and maintained in the 2 to 3 ranges (Devlin et al. 1999). Values of these two agents were totaled at the end of each shift by nursing personnel. Additional agents used for severe agitation or withdrawal was not be assessed.

Laboratory measurements: ABG analyzer (Model-ABL 80, Flex Basic, Brand-Radiometer, USA), Chest X-ray A/P view (ARDS diagnosis was radiologically confirmed by Radiologist)

Ventilator: AVEA TM (16448) and EVENT ventilators were used in this study.

Failure of modality: APRV or LOVT (V-SIMV) failure is defined as the inability to maintain a $PaO2 \geq 60$ mm Hg or a $PaCO2 < 60$ mm Hg and a $PH \leq 7.18$.

Informed written consent was taken from the patient's guardian after duly informing the procedure of treatment, anticipated result, possible advantages, disadvantages and complications considering all ethical issues. Confidentiality was maintained both verbally and documentary by using separate locker and computer password. Protocol was approved by ethical committee of Dhaka Medical College Hospital. Statistical analyses were carried out by using the Statistical Package for Social Sciences SPSS version 22.0 for Windows software. All data were presented in mean values mean±SD. The results were presented in tables, figures. Comparisons between groups were made with unpaired t-test, paired t-test and Chi square test. Observations were recorded as statistically significant if a p-value <0.05.

Results

A total of 48 patients were enrolled in this protocol and they were divided into two groups. Each group had 24 patients. Group-l and group-II were considered APRV and V-SIMV respectively. There were no differences in baseline demographics between APRV and V-SIMV (Table I). It was observed that 70.8% patients were male in group I and 62.5% in group II, ($p>0.05$). The mean height was found

63.33±1.6 inch in group I and 63.8±1.8 inch in group II. Mean weight was found 56.33±5.2 kg in group I and 56.9±5.8 kg in group II, mean BMI was found 29.2±36.8 kg/m^2 in group I and 30.0±41.3 kg/m^2 in group II, (p>0.05). Regarding the diagnosis, it was observed that post laparotomy (PGHCV) with sepsis was more common in both groups which was 25% (Table II). Measurement value of PaO_2 was increased in both groups but sharply increased in APRV which was significant ($p < 0.05$) (Figure I). On the contrary, value of $PaCO_2$ trended downward but more downward in APRV which was significant ($p < 0.05$) (Figure II). Percentage of oxygen saturation was augmented in both groups but there was reflecting measurable difference (Figure III). Oxygen requirement was sharply declined in both groups but sharply decreased in APRV which was significant ($p < 0.05$) (Figure IV). Peak inspiratory pressure was displayed in (Figure V). Although these values trended downward in both groups but in APRV mode peak pressure declined sharply. Mean airway pressure was increased in both groups, that was displayed in (Figure VI) these values trended upward in both groups but in APRV mode mean airway pressure declined sharply. PaO2/FiO2 ratios were compared throughout the analysis reflecting measurable difference in oxygenation. (Figure VII). Fentanyl and midazolam were totaled for each 24-hour period and are displayed in Table III for the first six days of the study period. Less sedation was required in APRV group that was statistically significant.

Table I: Distribution of the study patients by age, sex and physical information (N=48)

Characteristics	Group I(N=24)		Group II(N=24)		P-Value
	N	%	N	%	
Sex					
Male	17	70.83	15	62.5	0.54 ns
Female	7	29.17	9	37.5	
Age (years)					
11-20'	3	12.50	4	16.67	0.332 ns
21 - 30	6	25.00	8	33.33	
31 - 40	4	16.67	7	29.17	
41 - 50	11	45.83	5	20.83	
Mean±SD	35.38 ±10.78		32.38±10.42		
Physical information					
Height (inch)n	63.33±1.6		63.8±1.8		0.344 ns
Weight (kg)	56.0±5.2		56.9±5.8		0.574 ns
BMI (kg/m^2)	29.2±3.6		30.0±4.1		0.941 ns

Table II: Distribution of the study patients by diagnosis (n=48)

Diagnosis	Group I(N=24)		Group II(N=24)	
	N	%	N	%
Post laparotomy (PGHCV) with sepsis	6	25	6	25
Post laparotomy (Ileal perforation) with sepsis	3	12.5	0	0
Post laparotomy (stab injury on abdomen) with sepsis	0	0	2	8.33
Acute pancreatitis with septicemia with AKI with DM with sepsis	0	0	1	4.17
Carcinoma of colon (operated) with sepsis	1	4.17	0	0
Post laparotomy with sepsis	1	4.17	0	0
Post laparotomy due to blunt trauma on abdomen with sepsis	0	0	1	4.17
Post laparotomy(intestinal obstruction) with sepsis	1	4.17	0	0
Primary PPH with abdominal hysterectomy with sepsis	1	4.17	0	0
SLE with CNS involvement with sepsis	0	0	1	4.17
SLE with vasculitis with sepsis	1	4.17	0	0

Hemorrhaghic stroke with LSH with aspiration pneumonia	1	4.17	1	4.17
Post partam eclampsia with aspiration pneumonia	2	8.33	0	0
Post partam eclampsia with aspiration pneumonia	1	4.17	1	4.17
Meningoencephalitis with aspiration pneumonia	1	4.17	0	0
Epilepsy with aspiration pneumonia	1	4.17	1	4.17
GBS with aspiration pneumonia	0	0	1	4.17
Ischaemic stroke with RSH with aspiration pneumonia	1	4.17	0	0
OPC poisoing with aspiration pneumonia	0	0	1	4.17
SAH with aspiration pneumonia	1	4.17	0	0
Severe pneumonia	0	0	1	4.17
Head injury (ICH) due to RTA	0	0	2	8.33
Head injury due to RTA	0	0	1	4.17
Open communicated left tibia-fibula fracture with degloving injury due to RTA	1	4.17	0	0
Right tibia-fibula fracture with degloving injury due to RTA	0	0	1	4.17
PPH with massive blood transfusion	0	0	1	4.17
Repair of ruptured uterus with H/O of 3 LUCS with DIC	0	0	1	4.17
Post laparotomy (Rupture ectopic pregnancy) with DIC	0	0	1	4.17
Stab injury on chest(right)	1	4.17	0	0

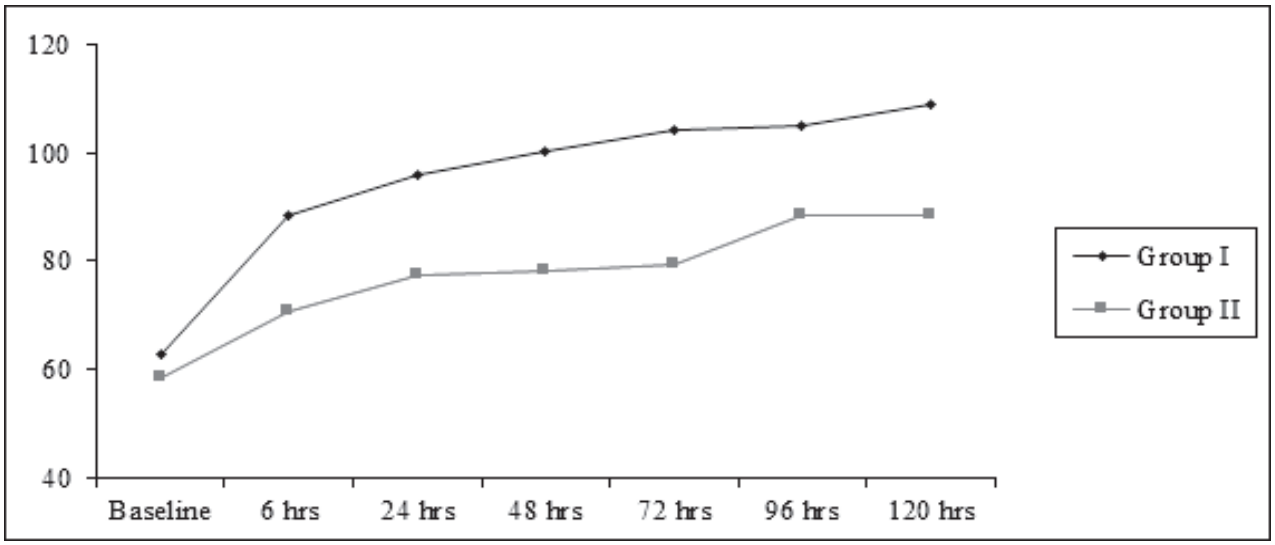

Figure I: PaO2 (mm Hg) values are greater for APRV patients than V-SIMV throughout the period

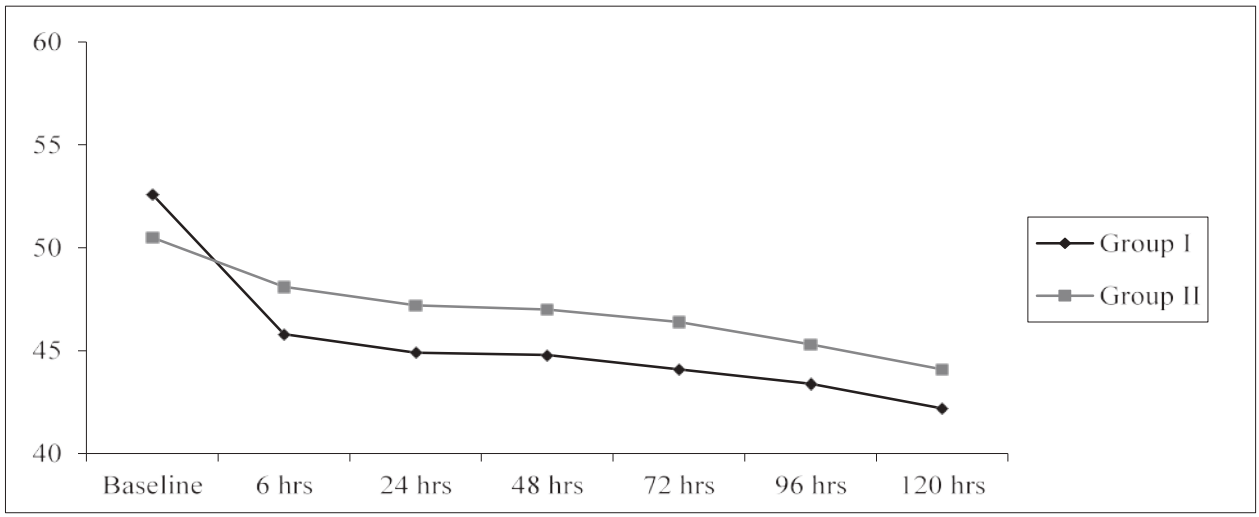

Figure II: $PaCO_2$ (mm Hg) differ in two groups in different time interval

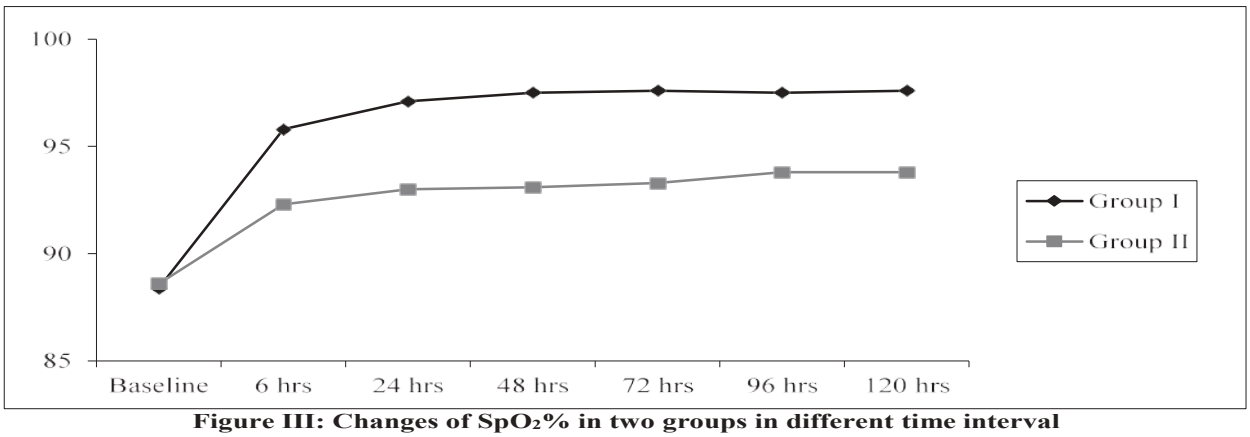

Figure III: Changes of SpO_2% in two groups in different time interval

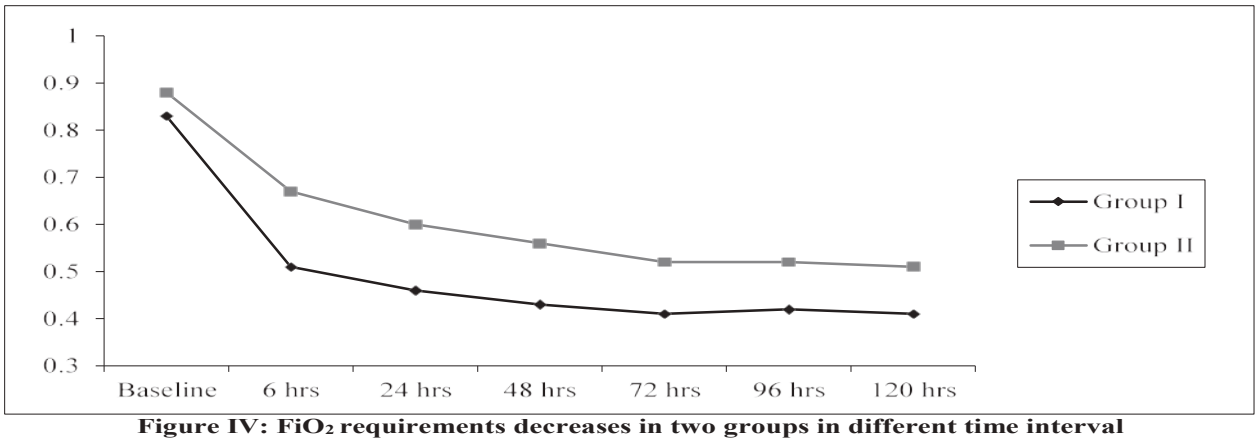

Figure IV: FiO_2 requirements decreases in two groups in different time interval

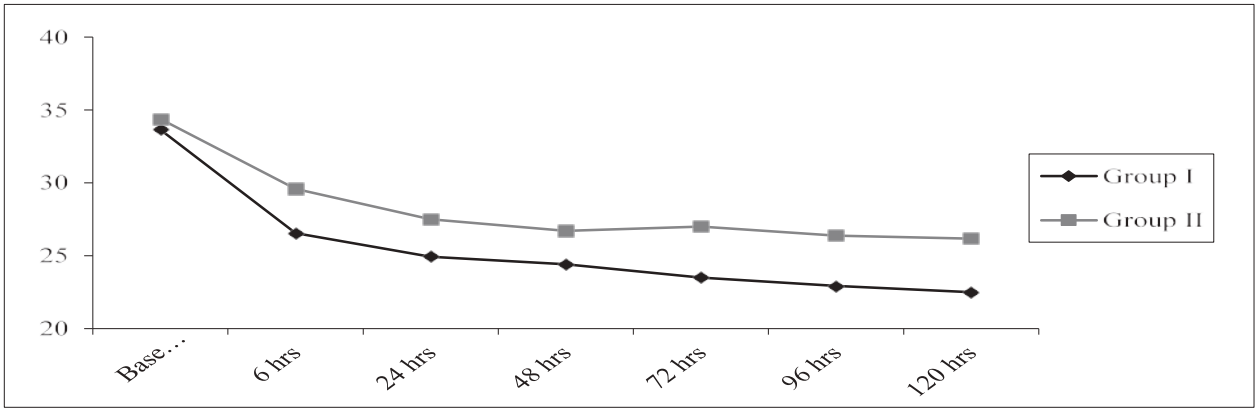

Figure V: Peak inspiratory pressure trended downward in two groups in different time intervals

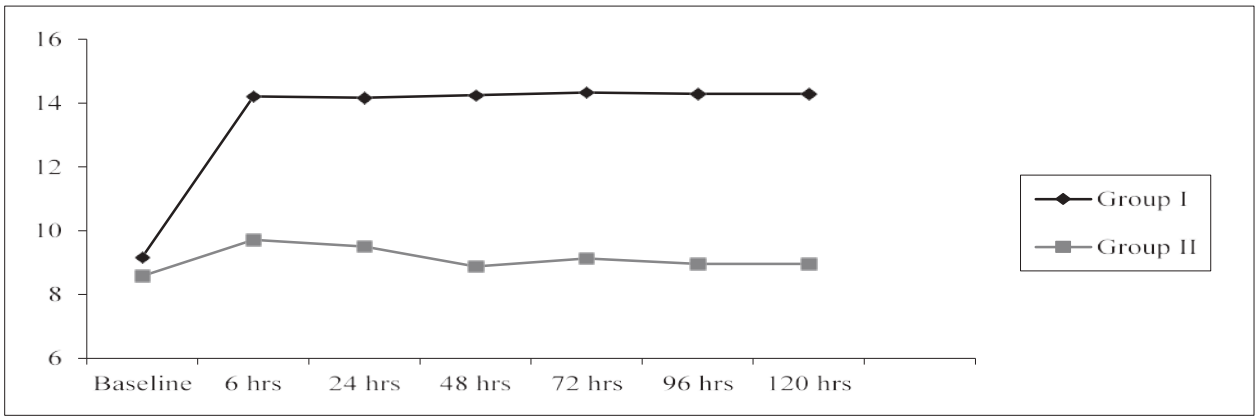

Figure VI: Mean airway pressure sharply increases in APRV patients

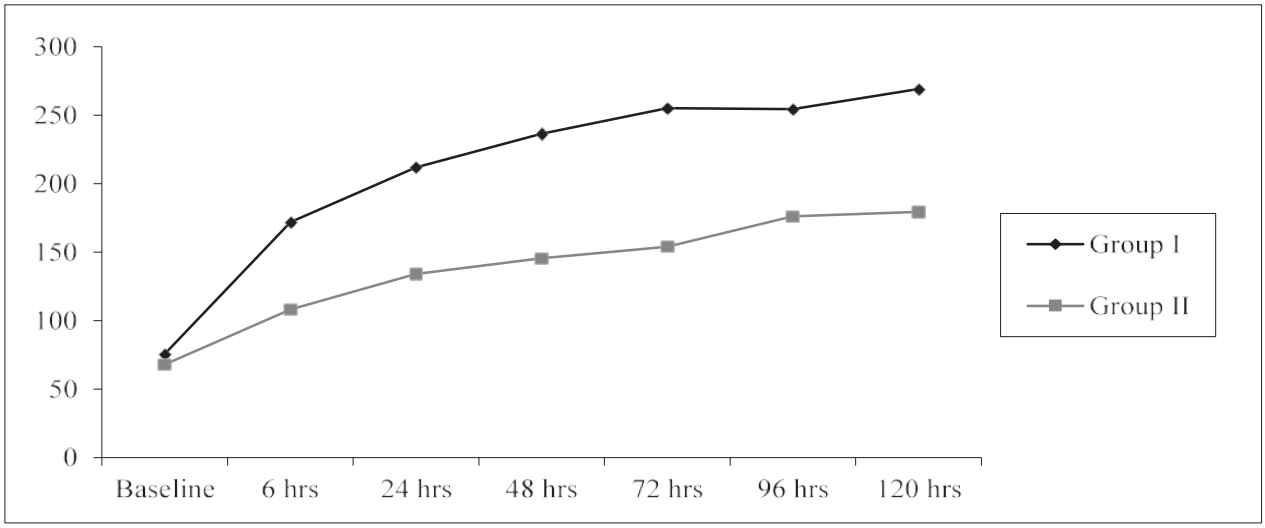

Figure VII: Changes of PaO_2/FiO_2 in two groups in different time interval

Table III: Sedation requirement in two groups in different time interval

Drugs	Group- I (n=24)		Group- II (n=24)		P value
	n	%	n	%	
Fentanyl(mcg)/ Midazolam(mg)					
24 hours					
500/45	10	41.7	24	100	0.001[s]
360/32.4	14	58.3	0	0	
48 hours					
500/45	4	16.7	20	83.3	0.001[s]
360/32.4	20	83.3	4	16.7	
72 hours					
500/45	3	12.5	20	83.3	0.001[s]
360/32.4	21	87.5	4	16.7	
96 hours					
500/45	2	8.3	15	62.5	0.001[s]
360/32.4	22	91.7	8	33.3	
120 hours					
500/45	1	4.2	9	37.5	0.001[s]
360/32.4	23	95.8	15	62.5	

Discussion

Although data were collected throughout the entire study period, a 6 - day period of observation was chosen for ventilator and blood gas values. This window of observation was chosen because repeated measures analysis of variance methodology eliminates all data from the data set for a patient once they were extubated. In this study, it was observed that almost half (45.9%) patients with acute respiratory distress syndrome belonged to age 41-50 years in group I and 20.8% in group II. The mean age was found 35.38±10.78 years varied from 18-50 years in group I and 32.38±10.42 years varied from 19-50 years in group II. The difference was almost similar between two groups. Maxwell et al.[17] found the mean age was found 40.5±14.1 years and 42.4±16.0 years in group I and group II respectively, which is comparable with the current study. On the other hand, some other studies found the which is higher with the current study[18-20]. The higher mean age and age range maybe due to geographical variations, racial, ethnic differences, genetic causes and different lifestyle may have significant influence on acute respiratory distress syndrome. In this present study it was observed that almost three fourth (70.8%) patients were male in group I and

(62.5%) in group II. Similarly, male predominant also observed by Luhr et al[21]. The mean height was found 63.33±1.6 inch in group I and 63.8±1.8 inch in group II. In our study the difference of height, weight and BMI was statistically not significant ($p>0.05$) between two groups. Regarding the diagnosis, it was observed that post laparotomy (PGHCV) with sepsis was more common in both groups which were (25.0%) and (25.0%) in group I and group II respectively. In this study, measurement value of PaO_2 was sharply increased with in first six hours in APRV patients. Rest of the period partial pressure of oxygen was increased in both groups but significantly higher in group I (APRV) in all follow-up. In contrast, Neumann et al.[28] using a porcine oleic acid lung injury model, found that PaO_2 was significantly increased using CPAP compared with APRV ($p<0.05$). The present data demonstrate that V-SIMV patients had higher $PaCO_2$. Functional residual capacity was not measured but the observed differences in gas exchange may reflect greater FRC in the APRV patients. In other words, increased mean airway pressure may in fact improve recruitment, which in turn would increase FRC and gas exchange. $PaCO_2$ is therefore reduced at lower levels of minute ventilation. In this study, it was observed that the mean PaO_2/FiO_2 was significantly increased from baseline at 6 hours later and subsequent follow-up up to 120 hours in both groups but significantly higher in group I (APRV) in all follow-up. Dart et al.[27] showed the PaO_2/FiO_2 ratio significantly increased (23%) after 72 hours of implementation of APRV in high-risk trauma patients. In this study, both decreased peak inspiratory pressures and increased mean airway pressure in APRV patients compared with control. Sydow et al.[32] also reported significantly decreased peak inspiratory pressures and increased mean airway pressures in 1994, well before the advent of lung protective strategy. Varpula et al.[33] have shown that, while controlling for mean airway pressure or plateau pressure, peak inspiratory pressure will decrease when converting to APRV from other modes of ventilation. Kaplan et al.[19] showed that both peak inspiratory pressure and mean airway pressure were reduced when patients were converted from pressure control ventilation to APRV when P_H was set at 75% of the peak pressure. Lower airway pressures also prevent excessive stretch and over distension of relatively normal lung segments[34]. Additionally, APRV allows spontaneous breathing to occur independently from the set ventilator cycle, which may allow recruitment of dependent lung areas adjacent to the heart and diaphragm[35]. In this study, it was observed that the mean SpO_2 was significantly increased from baseline at 6 hours later and subsequent follow-up up to 120 hours in both groups but significantly higher in group I in all follow-up. In a study Meade et al.[31] observed a positive response was an absolute increased in arterial oxygen saturation measured via pulse oximetry (SpO_2) of ≥3% within 5 min of completing the RM. Current study observed that the mean FiO_2 was significantly decreased in group I. Arterial desaturation, the hallmark of ARDS, can usually be improved to safe levels by increasing the fraction of inspired oxygen (FiO_2) and by applying positive end-expiratory pressure[36]. During this study no patient was expired from enrollment to 120 hours. Protocol -based sedation was used in present study. APRV patients required less sedative drugs. It is unclear why such variability of the mean daily sedation requirement occurred. Varpula et al.[22] reported no difference in propofol or fentanyl requirements between APRV and SIMV groups. Putensen et al.[23] showed decreased sulfentanil and midazolam use in APRV compared with pressure control ventilation patients for the first 72 hours of the study.

Limitations of the study: This was a prospective randomized controlled trial with a small sample size. So the findings of this study may not reflect the exact scenarios of whole country.

Conclusion And Recommendations

PaO_2, SpO_2, mean airway pressure and PaO_2/FiO_2 were significantly increased

(improved) in both groups but significantly higher in APRV group. $PaCO_2$, FiO_2 and peak inspiratory pressure were significantly decreased in both groups and significantly lesser in APRV group. Less sedation was required in APRV group. This study recommends use of APRV as a ventilator mode in ARDS patients. In this study FiO_2, doses of sedation and peak inspiratory pressure was decreased significantly in APRV mode. PaO_2 and mean airway pressure were significantly increased in APRV group than V-SIMV group. So, ICU physicians may use APRV mode in the management of ARDS patients. Further studies can be undertaken by inclusion of large number of patients with ARDS.

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Conflict of interest: None declared

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