Conventional Lung Protective Strategy Versus Airway Pressure Release Ventilation In Acute Lung Injury : Effects On Cardiac Performance And Lung Mechanics

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Abstract

Introduction: Since it's initial description lung protective strategy using tidal volumes of less than or equal to 6 ml/kg predicted body weight, with high respiratory rates and maintaining plateau pressures less than or equal to 30 cmH2O has been the standard ventilator management for ALI/ARDS patients, where a large number of randomized controlled clinical trials have proven it to reduce the mortality rates.

Airway pressure release ventilation (APRV) has been successfully used in neonatal, pediatric, and adult forms of respiratory failure. Experimental and clinical use of APRV has been shown to facilitate spontaneous breathing and is associated with decreased peak airway pressure and improved oxygenation[.

Methods: Our study was conducted on twenty patients admitted in the Critical Care Department, Cairo University Hospital with proven diagnosis of ALI/ARDS, who were ventilated sequentially for twelve hours using Volume Control – Assisted Controlled mechanical ventilation with lung protective strategy settings and Airway pressure release ventilation. Every three hours haemodynamic variables, arterial blood gases, lung mechanics and need for sedation were assessed. Aiming to compare and evaluate the two modes as regards haemodynamic effects, impact on arterial blood gases, need for sedation and the effects on respiratory mechanics.

Results: We did not demonstrate any significant change in the haemodynamic variables (heart rate, mean blood pressure, central venous pressure, and pulmonary capillary wedge pressure) between both modes of ventilation.(p-value>0.05) Partial pressure of carbon dioxide , acid-base status, and serum bicarbonate level did not change significantly between the two modes of mechanical ventilation (p-value>0.05),there was a significant decrease in partial pressure of oxygen with APRV compared to twelve hours of controlled mechanical ventilation with lung protective strategy with a 21.3% reduction (CMV 166.1±46.3, APRV 130.8±47.9) p-value 0.046, there was a highly significant 20.1% reduction in Hypoxic Index after twelve hours of APRV following twelve hours of CMV (CMV 275±80.7, APRV 219.9±86.8) p-value 0.001, there was a significant increase in respiratory rate and minute ventilation by 29.3% and 26.7% respectively during the twelve hours of APRV compared to twelve hours of CMV with respiratory rate increase from 21.5± 6.9 with CMV to 27.8± 6.2 APRV, p-value 0.007.There was no significant changes in dynamic compliance between both modes of ventilation (53±25.8 CMV, 52.9±26.9 APRV) p-value > 0.718. Highly significant decrease of 38.9% in peak airway pressure was noted during ventilation with APRV compared to CMV,(APRV 19.8±4, 32.4±6.7 CMV), p-value 0.0001, Associated with that there was also a highly significant 19% decrease in mean airway pressure during ventilation with APRV compared to CMV with lung protective strategy (CMV 18.9±3.7, APRV 15.3±3.3), p-value 0.0001. The need for sedation by propofol during the twelve hour period of APRV significantly decreased by 56.2% compared to the twelve hours of application of lung protective ventilation using CMV, where the dosage of propofol decreased from 1.6±0.5mg/kg/hr with CMV to 0.7±0.8mg/kg/hr with APRV, p-value 0.0001

Conclusion: APRV can be used safely as one of the optimum ventilatory strategies in patients with ALI/ARDS as it decreases airway pressures significantly and decreases the need for sedation while maintaining adequate oxygenation without altering haemodynamics.

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List of Abbreviation

AFC: Alveolar Fluid Clearance

ALI: Acute Lung Injury

APRV: Airway Pressure Release Ventilation

ARDS: Acute Respiratory Distress Syndrome

ARDSNet: Acute Respiratory Distress Syndrome Network

CO2: Carbon Dioxide

CPAP: Continuous Positive Airway Pressure

CT: Computed Tomography

EEL: End Expiratory Lung Volume

ERLV: End Release Lung Volume

FiO2: Fractional inspired oxygen

FRC: Functional Residual Capacity

HFOV: High Frequency Oscillatory Ventilation

HFV: High Frequency Ventilation

I:E: Inspiratory: Expiratory ratio

ICU: Intensive Care Unit

IL: Interleukin

INO: Inhaled Nitric Oxide

IVC: Inferior Vena Cava

MSP: Mean Systemic Pressure

MV: Minute Ventilation

NIH: National Institute of Health

NMBA: Neuromuscular Blocking Agents

NO: Nitric oxide

PaCO2: Partial pressure of arterial carbon dioxide

PaO2: Partial pressure of arterial oxygen

PBW: Predicted Body Weight

PEEP: Positive End Expiratory Pressure

PEFR: Peak Expiratory Flow Rate

Phigh: High set pressure

Plow: Low set pressure

Pmean: Mean airway pressure

PMN: Polymorphonuclear neutrophil

Ppeak: Peak airway pressure

Pplat: Plateau pressure of the airway

P-V: Pressure-Volume

RA: Right Atrium

RM: Recruitment Maneuvers

SIMV: Synchronized Intermittent Mandatory Ventilation

STC: Shock Trauma Center

TOP: Threshold Opening Pressure

TRALI: Transfusion Related Lung Injury

V/Q: Ventilation/Perfusion ratio

VAP: Ventilator Associated Pneumonia

VC-CMV: Volume control- Assisted control Mandatory Ventilation

VILI: Ventilator Induced Lung Injury

Vt: Tidal volume

vWF: Von Willebrand Factor

WOB: Work Of Breathing

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Introduction

Since its initial description in1967, several definitions have been proposed for the diagnosis of ARDS and for acute lung injury (ALI).At the present, the most commonly used definitions are those proposed by the American-European Consensus Conference[1]. ALI is defined as a "syndrome of inflammation and increasing permeability that is associated with a constellation of clinical, radiographic and physiologic abnormalities that cannot be explained by, but may coexist with, left atrial or pulmonary capillary hypertension."Based on the severity of hypoxemia, ARDS is defined as a severe form of ALI{i.e., an arterial partial pressure of oxygen/fraction of inspired oxygen (PaO_2/FiO_2)ratio less than 200 rather than 300 mmHg} [1].

ARDS and ALI are diffuse parenchymal injuries that have specific diagnostic criteria

- Acute onset disease
- PaO_2/FiO_2<200(ARDS)<300 mmHg(ALI)
- Bilateral lung infiltrates on chest radiograph
- Injury not reflecting cardiogenic pulmonary edema (PCWP <18mmHg) [2].

The overall goals in mechanical ventilatory support in parenchymal lung injury are to provide adequate gas exchange while minimizing any potentially iatrogenic lung injury. Although many variables can be monitored during this process, clinical decisions generally involve balancing four important factors: arterial pH, arterial hemoglobin saturation ($Sa0_2$), lung stretch, and lung exposure to oxygen [2].

- pH:7.20-7.45
- SaO_2:>88%
- P plat:<35 cmH_2O
- FiO_2:<0.6

The lung is subject to stretch injury in one of two ways: a shear stretch injury from repeated opening and closing of diseased alveoli and an overstretch injury induced by excessive distension at end inspiration. Addressing these two issues is the concept behind lung protective strategies [2].

In general, the first goal is to provide enough PEEP to recruit the recruitable alveoli while simultaneously not applying so much PEEP that healthier regions are over distended unnecessarily. The second goal is to avoid a PEEP/Tidal volume combination that unnecessarily over distends the lung at end inspiration. In summary, balancing these four clinical goals of pH, Sao_2, lung stretch, and FiO_2 constitutes the art of mechanical ventilation in parenchymal lung injury [1].

Generally, severe respiratory failure is managed during the acute phases with an assist/control (A/C) mode of ventilation. This ensures that all breathes have positive pressure supplied by the ventilator to provide virtually all the work of breathing. The assist capabilities of A/C ventilation allow the patient to trigger breaths. This may help in controlling CO_2 and improving patient comfort. If an inappropriate respiratory drive exists or patient triggering of assisted breaths is uncomfortable, sedation or paralysis or both may be needed such that only the control breaths of A/C ventilation are provided [2].

The tidal breath in volume controlled ventilation should be set in such a way that the plateau pressure is less that 35 cmH_2O, Tidal volume as low as 5 to 6 ml/kg may be needed [1].

A reasonable starting point is a normal frequency of between 12 to 20 breaths per minute .Increasing the frequency increases minute ventilation and generally increases CO_2 clearance .At some point; however, air trapping develops because of inadequate expiratory times. Setting the inspiratory time and the I: E ratio involves several considerations .The normal I: E ratio is roughly 1:2 to 1:4 [2].

Use of pressure-volume curves to set the PEEP/Tidal volume combination between the upper and lower inflection points. A modification of the conventional static approach uses very slow inspiratory flow and then measures upper and lower inflection points from the resulting dynamic pressure-volume curves. Gas exchange criteria to guide PEEP application involve several potential strategies. In general, commonly used "operational" ranges for PEEP in parenchymal lung injury are from 8 to 25 cm H_2O [2].

APRV is a ventilatory support pattern that provides a moderately high (i.e., 15 to 25 cm H_2O) level of continuous baseline airway pressure that is