Mechanical Ventilation in ARDS: Interaction of Respiratory Mechanics and Gas Exchange

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Introduction

Acute respiratory distress syndrome (ARDS), the most severe type of acute respiratory failure, was first described in the late 1960’s [1]. ARDS is the most severe manifestation of acute lung injury (ALI) that results from inflammation, endothelial injury and increased microvascular permeability. The definition of when ALI becomes ARDS is arbitrary, since the severity of the disease process is dynamic [2-4].

Mechanical ventilation is the basic life-saving supportive therapy in ARDS. It allows time for the lungs to heal from the primary insult, and has largely eliminated deaths in acute hypoxemia in the early phase of ARDS. Despite this, high pressures and overdistention of the lung, and shear stress due to repeated closure and reopening of alveoli during mechanical ventilation may markedly worsen the lung injury. Mechanical ventilation may also reduce the cardiac output and alter its distribution, and thereby indirectly contribute to the development of organ dysfunction and later death in ARDS [2, 5-7].

The main goal of mechanical ventilatory support should be to maintain gas exchange and sufficient tissue oxygenation. Simultaneously, the adverse effects of mechanical ventilation on hemodynamics, lung overdistention, and oxygen toxicity should be avoided or minimized. Several different modes of positive pressure ventilation are capable of supporting oxygenation and alveolar ventilation in ARDS [8-13]. None of the ventilatory modes have been shown superior to others in ARDS [2]. Accumulating data from experimental models and clinical studies strongly suggest that avoiding overinflation and high alveolar pressures should be one of the main goals in ALI. Furthermore, prevention of repeated collapse and reinflation of lung units by use of sufficient positive end-expiratory pressure (PEEP) may help to prevent further lung damage [5].

The mechanical and gas exchange characteristics of the respiratory system change dynamically during the course of ARDS. Optimum strategy for mechanical ventilatory support must therefore be frequently re-evaluated. The goal of this chapter is to review the physiological interaction between respiratory mechanics and ventilatory demand in ARDS. We also present a step-by-step approach for the selection of appropriate pressure, volume and arterial PCO$_2$-level in the clinical management of ARDS.
Gas Exchange and Respiratory Mechanics in ARDS

The early stage of ARDS is characterized by alveolar edema resulting from the endothelial injury and increased microvascular permeability. Aerated lung volume is markedly reduced: less than one half of the total lung volume may be aerated [14, 15]. Arterial hypoxemia due to increased venous admixture is the dominating gas exchange abnormality early in ARDS. Within days, impaired elimination of carbon dioxide due to an increased physiological dead space to tidal volume ratio becomes more and more important [16, 17]. This increases the ventilatory demand at any given level of arterial PCO$_2$. The ventilatory demand is further amplified by increased CO$_2$ production due to hypermetabolism. The gas exchange abnormalities are accompanied by the gradual impairment of the mechanical properties of the lung. The compliance of the lung decreases substantially and the resistance of both the airways and the lung tissue increases.

Mechanics and Ventilator Settings

The main effect of ALI on respiratory mechanics is a substantial reduction of compliance [14, 15, 18, 19]. Therefore, more inflation pressure is required to obtain a given tidal volume. The lung injury and impairment of respiratory mechanics is not homogeneous and the compliance in various parts of the lung vary from near normal to markedly reduced [14, 15]. The less injured areas with higher compliance will receive more of the tidal volume during inflation, and there is a substantial risk of overdistention and further injury. During expiration, parts of the lung may tend to collapse, and this can be prevented by the cautious use of PEEP [14, 15, 20].

Peak inspiratory pressure depends markedly on the inspiratory flow and the resistance of the ventilator tubing, intubation tube, and airway resistance, and is a poor indicator of alveolar pressure [19–21]. The plateau pressure at the end of an inspiratory pause is a better indicator of the alveolar pressure. The alveolar pressure at end-inspiration will be best obtained as the static pressure after 3 to 5 sec of end-inspiratory occlusion. This option is available in many modern ventilators.

Maximum safe level of alveolar pressure in ALI is not known. According to current recommendations, the static alveolar pressure during inflation should be limited to 30–35 cmH$_2$O [4]. The actual safe pressure levels are likely to vary substantially both between patients and over time in any patient. Estimation of the static compliance (Cst) is useful in tuning the ventilator [14, 15, 19–22].

$$Cst = \frac{\text{Tidal volume}}{\text{Pi, st} - \text{PEEPtot}}$$

where Pi, st is the static pressure after end-inspiratory occlusion (or pause pressure, if inspiratory occlusion is not available), and PEEPtot is the total PEEP obtained after end-expiratory occlusion, respectively. Though the basic physiological principles of interactions of respiratory mechanics and ventilator settings are not fully established, the changes in pressure-volume relationships for clini-