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Letter to Editor

Mechanical Ventilation and Ventilator Induced Lung Injury

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Artificial ventilation is an accepted concept in the modern therapeutic modalities. One of the most popular kinds of artificial ventilation is mechanical ventilation (MV) which is a supportive care base on respiratory pathophysiology [1]. Although, mechanical ventilation could save the patient life who suffers from respiratory failure but it could be potentially harmful if we have not a clear perception and strategy. MV could produce lung physiological and morphological alterations [1-3].

MV can trigger a cascade of events that eventually lead to a critical condition. Ventilator induced lung injury (VILI) is almost the collateral damage of the MV. Although VILI is an acute lung injury (ALI) developing during mechanical ventilation, it could be termed VILI if it is proven to be caused from artificial ventilation otherwise; it is termed Ventilator Associated Lung Injury (VALI). It is difficult to determine if the lung injury that a patient has developed was caused by the ventilatory set up or was because of the patient's worsening underlying lung condition. VALI is the appropriate term in most situations because it is practically impossible to confirm the real cause of injury. Neither VALI nor VILI need to be distinguished from progressive acute respiratory distress syndrome as their treatment strategy is the same [2,3].

VILI results from mechanical disruption of blood-gas barrier and consequent edema, release of inflammatory mediators and worsening hypoxemia. Alveolar injury results in increased capillary permeability, interstitial and alveolar swelling and haemorrhage, hyaline membranes defect, low surfactant content, cytokine release and diffused heterogenic lung injury [4,5].

Over distension and collapse are the principal initiators of alveolar injury during mechanical ventilation. Although high airway pressure in MV causes tearing of alveolar membrane and proposes barotrauma as the cause of VILI, not any high airway pressure causes lung injury. Musicians who play blowing instruments may develop very high airway pressures without experiencing barotraumas. Some researchers express that the triggering factor of VILI is not barotraumas. They make clinical relevance of high and low lung volume injury under suspicious and they believe the significance of inflammatory alterations observed during VILI is debated. However; it is well defined that VILI is the result of adverse interaction between MV and heterogeneity of lung tissue [3-5]. The size and function of alveoli are not the same, so they tolerate different pressure during mechanical ventilation. Lung is divided to 4 zones based on relationship between the pressure in the alveoli, in the arteries, in the veins and the pulmonary interstitial pressure and, all of which are affected by gravity [2,4].

It was propounded that positive end expiratory pressure (PEEP) can attenuate the effect of barotrauma and volutrauma. Although it is acceptable that physiologic PEEP reduces VILI, and it was attributed to reduction of cyclic opening and closing of alveoli in a routine ventilator setup; the effects of applied high PEEP strictly depend on lung capacity, which varies widely based on lung compliance. The

protective effect of physiologic PEEP in a normal lung is the result of limiting dynamic strain due to tidal volume at the cost of static strain and attenuation of stress risers. Applying increased PEEP may lead to opposing effects on a main factor potentially worsening the lung injury [4,5].

The lung injury due to alveolar distension is referred to alveolar strain. It reflects the presence of elevated trans-pulmonary pressure (TPP). A TPP of 17 cmH_2O increases lung volume to its anatomical limit which is predisposing to VILI [3,4].

Viscoelastic property of lung makes pulmonary mechanics dependent on time so that stress increases in elevated respiratory rate or decreased inspiratory time. Physiologic alveolar heterogeneity acts as a stress riser and multiplying global stress at regional level. Limitation of stress rather than strain (alveolar end tidal volume) is the safe strategy of mechanical ventilation to prevent VILI. Driving pressure is the non-invasive surrogate of lung strain, but its relation to alveolar plateau pressure dependents on the alveolar dynamic compliance [3,5,6].

In summary, VILI is an ALI affecting the respiratory tract and lung parenchyma that is caused by MV. Tidal volume or induced driving pressure, positive end expiratory pressure and; inspiratory time and flow play the main role in adjusting ventilator setup for protection or management of VILI.

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