History and development of ARDS

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Ashbaugh et al. (1967) proposed the first ARDS description, based on refractory hypoxemia, bilateral pulmonary infiltrates (X-ray), low respiratory system compliance, increased lung weight. In the years other definitions have been introduced, being the most widely accepted one the definition proposed by the American-European Consensus Conference which relies primarily on PaO2/FiO2 < 300 and bilateral infiltrates. New insights were provided by the CT-scan over the last two decades, demonstrating that ARDS does not homogeneously affect the lung parenchyma. The lung injury severity is widely distributed in ARDS population (5 to 70% of the total lung weight) and it is strictly associated with the severity of injury: greater is the amount of gasless tissue at 5 cmH2O PEEP, greater is the amount of gasless tissue regaining aeration at 45 cmH2O airway pressure.

The way of ventilating the ARDS lung deeply changed over the last 30 years, primarily due to a better understanding of physiological mechanisms involved in the mechanical ventilation, as heart and lung interaction and generation of ventilator induced lung injury. In the new century, beginning with the National Institute of Health NIH low tidal volume ventilation, a series of therapeutical approaches have been proposed and tested in ARDS, some successful, as prone position and artificial lung support in severe ARDS, some unsuccessful, as high frequency ventilation, and some still questionable and debated, as the use of higher PEEP compared to lower PEEP. At the same time, the mechanisms of ventilation induced lung injury, the primary risk of mechanical ventilation in ARDS, have been furtherly investigated both in its physical and biological components. Recently this bulk of knowledge has been embedded in the Berlin ARDS definition, which pragmatically classifies the degree of severity of the syndrome and, more important, suggests possible treatments scaled to the severity. It is not clear, however, within a certain degree of severity which criteria should guide the possible alternative treatment. As an example, in severe ARDS, prone position, extracorporeal oxygenation should be applied separately or in combination? And, more important, should these techniques be available in every hospital or concentrated in referral centers?

ARDS and VILI

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Stress and strain are the primary determinants of ventilator-induced lung injury. Nowadays, in clinical practice, their surrogates are considered airway pressure and tidal volume normalized for ideal body weight Prevention of ventilator-induced lung injury is primarily based on recognizing the "harmful" threshold for these surrogates (30 cm H₂O airway plateau pressure and 6 ml/kg VT IBW). However and airway plateau pressure and tidal volume normalized for ideal body weight are poor surrogates for lung stress and strain as the real distending force of the lung is the transpulmonary pressure and the rough equivalent of lung strain is the ratio of tidal volume to functional residual capacity. Experimental studies on pigs investigated a possible strain threshold and other studies are ongoing trying to identify the effects of the single ventilatory parameters on VILI. In healthy lungs, ventilation becomes lethal with strain reaching total lung. The striking discrepancy between experimental data and clinical scenarios (harm at VT of 12 mL/kg) may be explained by lung unhomogeneity, locally generating "stress risers" or "pressure multipliers". When mechanical ventilation becomes unsafe artificial lungs may provide adequate gas exchange while reducing the load of mechanical ventilation.