Ventilator induced lung injury

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摘要

Abstract

During mechanical ventilation, high end-inspiratory lung volume (whether it be because of large tidal volume (VT) and/or high levels of positive end-expiratory pressure) results in a permeability type pulmonary oedema, called ventilator-induced lung injury (VILI). Previous injury sensitises lung to mechanical ventilation. This experimental concept has recently received a resounding clinical illustration after a 22% reduction of mortality was observed in acute respiratory distress syndrome patients whose VT had been reduced. In addition, it has been suggested that repetitive opening and closing of distal units at low lung volume could induce lung injury but this notion has been challenged both conceptually and clinically after the negative results of the Acute Respiratory Distress Syndrome clinical Network Assessment of Low tidal Volume and Elevated end-expiratory volume to Obviate Lung Injury (ARDSNet ALVEOLI) study. Experimentally and clinically, involvement of inflammatory cytokines in VILI has not been unequivocally demonstrated. Cellular response to mechanical stretch has been increasingly investigated, both on the epithelial and the endothelial side. Lipid membrane trafficking has been thought to be a means by which cells respond to stress failure. Alterations in the respiratory system pressure/volume curve during ventilator-induced lung injury that include decrease in compliance and position of the upper inflection point are due to distal obstruction of airways that reduce aerated lung volume. Information from this curve could help avoid potentially harmful excessive tidal volume reduction.