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TIDAL VOLUME AND VENTILATION DURATION MODULATE LUNG INJURY IN EXPERIMENTAL ACUTE RESPIRATORY DISTRESS SYNDROME DESPITE SIMILAR CUMULATIVE MECHANICAL POWER

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BACKGROUND: Mechanical ventilation is essential in ARDS management but may cause ventilator-induced lung injury (VILI) when excessive mechanical power is delivered. While safe power thresholds have been proposed, the role of ventilation duration—particularly under equivalent cumulative mechanical power—remains unclear. We hypothesized that tidal volume (VT) and ventilation time independently influence VILI severity, even when total power exposure is matched. OBJECTIVES: To evaluate how different combinations of VT and ventilation duration, matched for cumulative mechanical power, affect lung mechanics, histological injury, and molecular biomarkers in an experimental ARDS model. INTERVENTIONS: ARDS was induced via intratracheal Escherichia coli lipopolysaccharide. After 24 hours, animals were ventilated (PEEP = 3 cmH_2O) using three VT–duration strategies with matched cumulative mechanical power: 1) low VT (6 mL/kg) for 150 min (LVT-HMV), 2) moderate VT (9 mL/kg) for 100 min (MVT-MMV), and 3) high VT (12 mL/kg) for 75 min (HVT-LMV). An LPS-exposed, non-ventilated group served as molecular control. RESULTS: Despite similar cumulative mechanical power across groups—LVT-HMV: 48.4 (44.4–56.7), MVT-MMV: 48.3 (44.2–55.4), HVT-LMV: 44.3 (42.1–55.4) J/min—the MVT- MMV group showed higher plateau and driving pressures, mechanical power, and lung overdistension. Markers of inflammation (IL-6), epithelial activation (amphiregulin), and extracellular matrix disruption (syndecan-1, versican) were elevated in MVT-MMV versus LVT-HMV. The HVT-LMV group showed more alveolar collapse, pulmonary edema, and upregulated IL-6 and VCAM-1, indicating endothelial injury. CONCLUSIONS: Tidal volume and ventilation time exerted distinct and additive effects on lung injury, independent of cumulative mechanical power. These findings challenge the adequacy of cumulative power as a sole predictor of VILI and highlight the need to optimize both volume and duration in protective ventilation strategies.

Palavras-chave: acute respiratory distress syndrome, mechanical power, tidal volume, inflammation, overdistension.