Preventing ventilator-induced lung injury

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BACKGROUND: Mechanical ventilation is commonly used in Intensive Care Units for supplying supplementary oxygen to critically ill people. However, mechanical ventilation itself often damages the recipient's lungs. This damage is termed ventilator-induced lung injury (VILI) and is associated with poor clinical outcomes. There are currently no effective pharmacological treatments for VILI prevention or treatment in routine clinical use.

METHOD: This study investigated 2 drugs aimed at therapeutic targets in mechanically ventilated lungs using an isolated perfused rat lung preparation (Hugo Sachs Elektronik/Harvard Apparatus IPL-2). Respiratory parameters were recorded using ADInstruments PowerLab and LabChart software. A hyperbaric model of VILI was developed. Lungs were maintained at normal tidal volume for 10 min using positive pressure ventilation (between +3 to +15 cmH_2O), and then hyperinflated by increasing the peak end-inspiratory pressure to +30 cmH_2O for a period of 1 h. After this, a 90 min measurement period was undertaken at normal negative breathing pressures (-2 to -12 cmH_2O). In treatment experiments (n=7), the drug was added to the recirculating perfusate 7 min prior to lung hyperinflation. Findings were compared to determine whether drug treatment reduced the severity of VILI.

RESULTS: The VILI model optimisation was successful, with hyperinflation resulting in an increase in tidal volume from 2.0 to 4.8 mL and a corresponding steady rise in lung weight by 16% associated with visible oedema in the lower lung lobes. The abnormal increased lung weight was sustained over the following 90 min normal ventilation period as well.

In the drug treatment group the weight gain and tissue oedema were significantly less severe in the hyperinflation injury period and normal ventilation periods after drug treatment.

CONCLUSION: It was found possible to pharmacologically attenuate the severity of experimental VILI using biochemical methods.

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