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Mild hypothermia increases pulmonary anti-inflammatory response during protective mechanical ventilation in a piglet model of acute lung injury.

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Abstract

BACKGROUND: The effects of mild hypothermia (HT) on acute lung injury (ALI) are unknown in species with metabolic rate similar to that of humans, receiving protective mechanical ventilation (MV). We hypothesized that mild hypothermia would attenuate pulmonary and systemic inflammatory responses in piglets with ALI managed with a protective MV.

METHODS: Acute lung injury (ALI) was induced with surfactant deactivation in 38 piglets. The animals were then ventilated with low tidal volume, moderate positive end-expiratory pressure (PEEP), and permissive hypercapnia throughout the experiment. Subjects were randomized to HT (33.5°C) or normothermia (37°C) groups over 4 h. Plasma and tissue cytokines, tissue apoptosis, lung mechanics, pulmonary vascular permeability, hemodynamic, and coagulation were evaluated.

RESULTS: Lung interleukin-10 concentrations were higher in subjects that underwent HT after ALI induction than in those that maintained normothermia. No difference was found in other systemic and tissue cytokines. HT did not induce lung or kidney tissue apoptosis or influence lung mechanics or markers of pulmonary vascular permeability. Heart rate, cardiac output, oxygen uptake, and delivery were significantly lower in subjects that underwent HT, but no difference in arterial lactate, central venous oxygen saturation, and coagulation test was observed.

CONCLUSIONS: Mild hypothermia induced a local anti-inflammatory response in the lungs, without affecting lung function or coagulation, in this piglet model of ALI. The HT group had lower cardiac output without signs of global dysoxia, suggesting an adaptation to the decrease in oxygen uptake and delivery. Studies are needed to determine the therapeutic role of HT in ALI.