Inactivation of hypoxic pulmonary vasoconstriction results in acute increase in extravascular lung water measurement by PiCCO in canine acute lung injury

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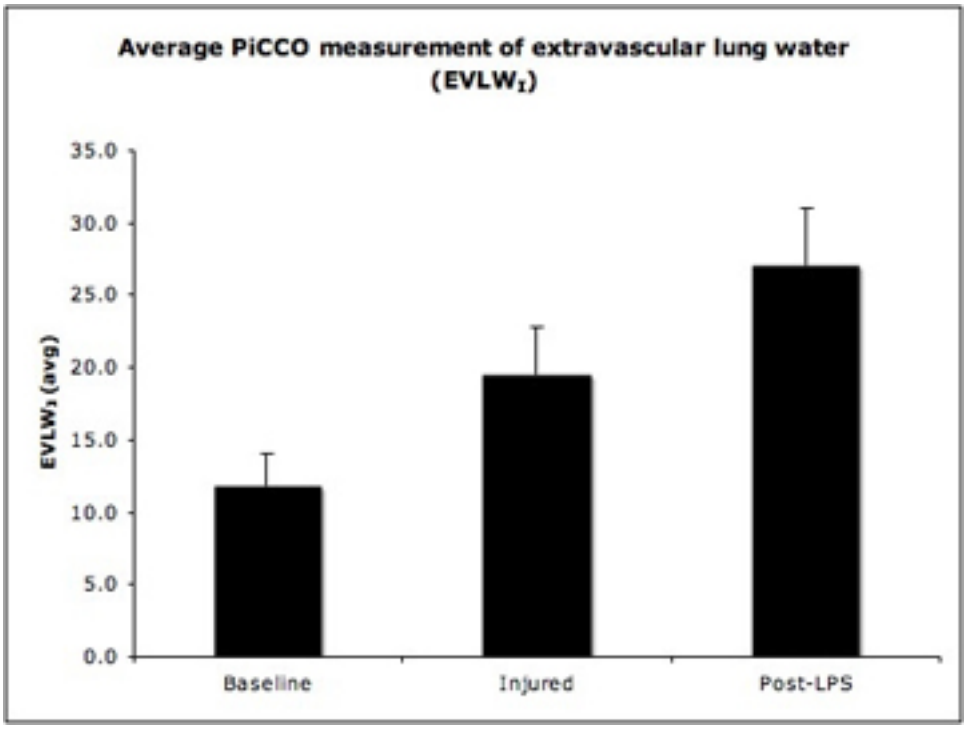
Introduction: Human and animal studies using the PiCCO transpulmonary thermodilution system (Pulsion, Germany) have demonstrated increased extravascular lung water (EVLW) measurements in the setting of worsening P/F ratios in sepsis and acute lung injury (ALI), attributed to progression of lung edema and flooding. However, we hypothesize that redistribution of blood flow resulting from the inactivation of hypoxic pulmonary vasoconstriction (HPV) is another possible cause.

Methods: 5 anesthetized, mechanically ventilated canine had PA and PiCCO catheters placed and lung injury induced with i.v. oleic acid (OA). Data including blood gases, hemodynamics, and PiCCO values were recorded every 30 minutes. Once P/F ratios decreased <300 and shunt fraction >20%, 15 mcg/kg i.v. endotoxin (LPS) was administered to inactivate HPV.

Results: OA injury caused an increase in peak and plateau pressures, slight fall in cardiac output (CO) and moderate pulmonary hypertension. LPS administration further decreased CO and dramatically decreased P/F and increased shunt. CO demonstrated good correlation between the PiCCO and PA catheter values (R^2= 0.90). EVLW increased gradually with OA injury and then markedly following LPS administration (Fig 1).



Conclusions: The abrupt increase in EVLW and shunt after LPS administration is consistent with inactivation of HPV and increased perfusion to already flooded lung regions that were previously

thermally silent. Future, studies will correlate these PiCCO measurements of EVLW with CT measures of lung tissue density and regional blood flow.

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