44620 - EFFECT OF CPB AND ANEMIA ON RAT RENAL TISSUE PO₂ AND eNOS EXPRESSION

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INTRODUCTION: Renal failure is a serious complication of cardiopulmonary bypass (CPB) surgery, with significant morbidity and mortality¹. Anemia is an independent predictor of RF following CPB², possibly due to inadequate renal oxygen delivery. We investigated the effects of CPB and anemia on renal cortical and medullary tissue oxygen tension (pO₂), blood flow (RBF) and eNOS protein expression.

METHODS: With Animal Care Committee approval, anesthetized rats (ketamine, isoflurane, fentanyl, propofol) underwent 1h of normothermic CPB. Two groups of animals were studied in which the target hemoglobin concentration (Hb) was either 100g/L (CPB) or 65g/L (CPB-Anemia). Combined oxygen sensing microelectrodes and laser Doppler flow probes (Oxford Optronix) were placed in the renal cortex and medulla in each rat for simultaneous measurement of RBF and pO₂. Absolute pO₂ values (mmHg) or relative increases in RBF (%) were measured. Different groups of rats were sacrificed immediately after bypass, or 6h post-bypass, to assess renal eNOS expression by immunohistochemistry. Statistical analysis was performed using ANOVA on ranks and Mann-Whitney rank sum tests.

RESULTS: There were no differences in physiologic variables between groups at baseline. Hb decreased from a baseline of 116±17 to 97±7 in the CPB group, and further decreased to 64±7 in the CPB-Anemia group (p<0.05). Basal renal cortical pO₂ values (16.1±9.2mmHg) were higher than medullary pO₂ values (4.9±3.0mmHg, n=8, p<0.001). Renal cortical pO₂ decreased during CPB (7.0±3.6, n=4) and CPB-Anemia (6.9±5.2, n=4) (p=0.031). Renal medullary pO₂ was decreased during CPB (2.7±1.7, n=4) and further decreased with CPB-Anemia (1.2±0.9, n=4) (p=0.015). Renal cortical and medullary blood flow did not change significantly during CPB but increased during CPB-Anemia (151±50% and 221±92%, n=8, p<0.05). Immunohistochemical analysis demonstrated eNOS staining in renal blood vessels immediately after CPB and increased eNOS staining 6 hours post-CPB in both groups (n=4).

DISCUSSION: In this model, tissue pO₂ was significantly lower in renal medulla compared with cortex prior to CPB. During CPB, pO₂ was reduced in both renal cortex and medulla with maintained RBF. CPB with anemia caused a further reduction in renal medullary pO₂, despite a significant increase in medullary blood flow. These data suggest that the renal medulla is more susceptible to hypoxic injury during CPB with anemia. Increased renal eNOS expression may play a role in mediating RBF during CPB and anemia.

REFERENCES:

1. Br J Anesth 2005 95:20-32

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