INTRODUCTION: The relationships between systemic hemodynamics and renal blood flow and renal microcirculation are poorly known in sepsis. Norepinephrine (NE) infusion may add another level of complexity. METHODS: Ventilated and anesthetized rats were submitted to various mean arterial pressure (MAP) steps by blood removal, in presence and absence of sepsis and/or NE. Renal blood flow (RBF) and blood velocity (Vm) in renal cortical capillaries (using Sidestream Dark Field Imaging) were measured. Data were analyzed using linear mixed models enabling us to display the effects of both the considered explanatory variables and their interactions. RESULTS: Positive correlations were found between MAP and RBF. Sepsis had no independent impact on RBF whereas norepinephrine decreased RBF, regardless of the presence of sepsis. The relationship between MAP and RBF was weaker above a MAP of 100 mmHg as opposed to below 100 mmHg, with RBF displaying a relative "plateau" above this threshold. Sepsis and NE impacted carotid blood flow (CBF) differently compared to RBF, demonstrating organ specificity. A positive relationship was observed between MAP and Vm. Sepsis increased Vm while nNE decreased Vm irrespective of MAP. Sepsis was associated with an increase in serum creatinine determined at the end of the experiments, which was prevented by NE infusion. CONCLUSION: In our model, sepsis at an early phase did not impact RBF over a large range of MAP. NE elicited a renal vasoconstrictive effect. Autoregulation of RBF appeared conserved in sepsis. Conversely, sepsis was associated with "hypervelocity" of blood flow in cortical peritubular capillaries reversed by NE infusion.
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