

Redistribution of intestinal microcirculatory oxygenation during acute hemodilution in pigs.

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Acute normovolemic hemodilution (ANH) compromises intestinal microcirculatory oxygenation; however, the underlying mechanisms are incompletely understood. We hypothesized that contributors herein include redistribution of oxygen away from the intestines and shunting of oxygen within the intestines. The latter may be due to the impaired ability of erythrocytes to off-load oxygen within the microcirculation, thus yielding low tissue/plasma $Po(2)$ but elevated microcirculatory hemoglobin oxygen ($HbO(2)$) saturations. Alternatively, oxygen shunting may also be due to reduced erythrocyte deformability, hindering the ability of erythrocytes to enter capillaries. Anesthetized pigs underwent ANH (20, 40, 60, and 90 ml/kg hydroxyethyl starch; ANH group: $n = 10$; controls: $n = 5$). We measured systemic and mesenteric perfusion. Microvascular intestinal oxygenation was measured independently by remission spectrophotometry [microcirculatory $HbO(2)$ saturation ($\mu HbO(2)$)] and palladium-porphyrin phosphorescence quenching [microcirculatory oxygen pressure in plasma/tissue ($\mu Po(2)$)]. Microcirculatory oxygen shunting was assessed as the disparity between mucosal and mesenteric venous $HbO(2)$ saturation ($HbO(2)$ -gap). Erythrocyte deformability was measured as shear stress-induced cell elongation (LORCA diffractometer). ANH reduced hemoglobin concentration from 8.1 to 2.2 g/dl. Relative mesenteric perfusion decreased (decreased mesenteric/systemic perfusion fraction). A paralleled reduction occurred in mucosal $\mu HbO(2)$ (68 ± 2 to $41 \pm 3\%$) and $\mu Po(2)$ (28 ± 1 to 17 ± 1 Torr). Thus the proposed constellation indicative for oxygen off-load deficits (sustained $\mu HbO(2)$ at decreased $\mu Po(2)$) did not develop. A twofold increase in the $HbO(2)$ -gap indicated increasing intestinal microcirculatory oxygen shunting. Significant impairment in erythrocyte deformability developed during ANH. We conclude that reduced intestinal oxygenation during ANH is, in addition to redistribution of oxygen delivery away from the intestines, associated with oxygen shunting within the intestines. This shunting appears to be not primarily caused by oxygen off-load deficit but rather by oxygen/erythrocytes bypassing capillaries, wherein a potential contributor is impaired erythrocyte deformability.

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