Crit Care Med. 2004 Jan;32(1):150-6.

Dopamine under alpha1-blockade, but not dopamine alone or fenoldopam, increases depressed gastric mucosal oxygenation.

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OBJECTIVE: To compare the effects of dopamine, both in the presence and absence of alpha1-blockade, and fenoldopam on microvascular gastric mucosal oxygenation and systemic oxygen transport under compromised circulatory conditions, both without and with fluid resuscitation. DESIGN: Randomized controlled animal study. SETTING: University department of anesthesiology. SUBJECTS: Eight anesthetized dogs with chronically implanted ultrasound flow probes around the pulmonary artery for continuous measurement of cardiac output. INTERVENTIONS: On different days, the dogs received in random order either dopamine (2.5 and 5.0 microg.kg(-1).min(-1), with or without alpha1-blocker pretreatment), the selective DA1-agonist fenoldopam (0.1 and 1.0 microg.kg(-1).min(-1), with and without DA1-blocker pretreatment), or saline (control). These interventions were performed under compromised cardiocirculatory conditions (induced by ventilation with positive end-expiratory pressure [PEEP] of 10 cm H2O), both without and with fluid resuscitation. MEASUREMENTS AND MAIN RESULTS: We continuously measured regional microvascular hemoglobin saturation (microHbO2) in gastric mucosa by reflectance spectrophotometry and systemic oxygen transport ([U1E0A]O2). Ventilation with PEEP significantly decreased [U1E0A]O2 (from 19 +/- 2 to 9 +/- 1 mL.kg(-1).min(-1), mean +/- sem) and gastric mucosal microHbO2 (from 57 +/- 2% to 37 +/- 3%). Fluid resuscitation restored [U1E0A]O2 back to baseline (from 9 +/- 1 to 19 +/- 2 mL.kg(-1).min(-1)) but only partially restored microHbO2 (from 37 +/- 3% to 50 +/- 4%). Under both conditions, dopamine with and without alpha1-blockade significantly increased [U1E0A]O2 (by about 5 mL.kg-1.min-1 in the nonresuscitated state and 10 mL.kg-1.min-1 in the fluid resuscitated state, respectively), but only dopamine in the presence of alpha1-blockade also significantly increased gastric mucosal microHbO2 (by 5 +/- 1% and 7 +/- 2% in the nonresuscitated and fluid resuscitated states, respectively). Fenoldopam under all study conditions did not significantly affect [U1E0A]O2 or microHbO2, either in the presence or absence of DA1-blockade. CONCLUSIONS: During compromised cardiocirculatory conditions, alpha1-receptor activation during dopamine infusion prevented an increase in gastric mucosal oxygenation. Furthermore, selective DA1-stimulation (by fenoldopam) was insufficient to overcome the PEEP-induced depression of microHbO2. The responses of gastric mucosal oxygenation did not parallel changes in systemic oxygen transport. These findings were independent of fluid resuscitation.

PMID: 14707574 [PubMed - indexed for MEDLINE]