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Effects of thoracic epidural anaesthesia on microvascular gastric mucosal oxygenation in physiological and compromised circulatory conditions in dogs{dagger}

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BACKGROUND: The effects of thoracic epidural anaesthesia (TEA) on gastric mucosal microvascular haemoglobin oxygenation (micro HbO2) are unclear. At the splanchnic level, reduction of sympathetic tone may promote vasodilation and increase micro HbO2. However, these splanchnic effects are counteracted by systemic effects of TEA (e.g., decreased cardiac output (CO) and mean arterial pressure (MAP)), thus making the net effect on micro HbO2 difficult to predict. In this respect, effects of TEA on micro HbO2 may differ between physiological and compromised circulatory conditions, and additionally may depend on adequate fluid resuscitation. Furthermore, TEA may alter the relationship between regional micro HbO2 and systemic oxygen-transport (DO2). METHODS: Chronically instrumented dogs (flow probes for CO measurement) were anaesthetized, their lungs ventilated and randomly received TEA with lidocaine (n=6) or epidural saline (controls, n=6). Animals were studied under physiological and compromised circulatory conditions (PEEP 10 cm H2O), both with and without fluid resuscitation. We measured gastric mucosal micro HbO2 by reflectance spectrophotometry, systemic DO2, and systemic haemodynamics (CO, MAP). RESULTS: Under physiological conditions, TEA preserved micro HbO2 (47 (3)% and 49 (5)%, mean (SEM)) despite significantly decreasing DO2 (11.3 (0.8) to 10.0 (0.7) ml kg(-1) min(-1)) and MAP (66 (2) to 59 (3) mm Hg). However, during compromised circulatory conditions, TEA aggravated the reduction in micro HbO2 (to 32 (1)%), DO2 (to 6.7 (0.8) ml kg(-1) min(-1)) and MAP (to 52 (4) mm Hg), compared with controls. During TEA, fluid resuscitation completely restored these variables. TEA preserved the correlation between micro HbO2 and DO2, compared with controls. CONCLUSIONS: TEA maintains micro HbO2 under physiological conditions, but aggravates the reduction of micro HbO2 induced by cardiocirculatory depression, thereby preserving the relationship between gastric mucosal and systemic oxygenation.

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