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Hypercapnia increases gastric mucosal oxygenation during hemorrhagic shock

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Objective:

Permissive hypercapnia (PHC), e.g., as component of a lung-protective ventilation mode [1], leads to systemic and regional vasodilatation [1]. It is unclear, whether this vasodilatation also increases gastrointestinal microvascular mucosal oxygenation (µHbO2). This should be beneficial in hemorrhagic shock, since adequate µHbO2 appears crucial to maintain an intact mucosal barrier [2]. Therefore, we tested the effects of PHC on µHbO2 during hemorrhagic shock.

Material & Method:

In anesthetized (1.5 MAC sevoflurane), ventilated dogs we measured µHbO2 of the gastric mucosa [3] (tissue spectrophotometry) and arterial lactate levels. The dogs were randomized to: PHC (etCO2=70 mmHg) with shock (n=6), normocapnia (etCO2=35 mmHg) with shock (n=6), and PHC without shock (n=6). Hemorrhagic shock was induced by acute withdrawal of 20% of blood volume. Statistics: Analysis of variance, Fisher´s PLSD, $p<0,05$.

Results:

PHC under baseline conditions (no shock) significantly increased regional mucosal oxygenation (µHbO2 from 53 ± 3 to 60 ± 2%) at a decreasing arterial lactate level. During shock, µHbO2 decreased significantly less under PHC (minus 3 ± 2%), compared to normocapnia (minus 14 ± 4%), and under PHC the arterial lactate increased during shock significantly less than under normocapnia (1.3 ± 0.1 vs. 2.2 ± 0.1 mmol/l). A PHC without shock demonstrated, that µHbO2 remains increased for hours and returns to baseline after normalisation of etCO2 to baseline.

Discussion:

PHC increases the gastrointestinal mucosal oxygenation. Since during shock the µHbO2 dropped significantly less under PHC than under normocapnia, a PHC may be a prophylactic/therapeutic option not only to protect the lung, but also to protect the gastrointestinal tract (i.e., the gastrointestinal mucosa).

Literatur:

[1] Laffey JG, Kavanagh BP: Lancet 1999;354:1283-6 [2] Sato N, Kamada T, Shichiri M, Kawano S, Abe H, Hagihara B: Gastroenterology 1979;76:814-9 [3] Schwarte LA, Picker O, Schindler AW, Fournell A, Scheeren TWL: Crit Care Med 2003;31:1999-2005