Effects of hydrogen sulfide on hemodynamics, inflammatory response and oxidative stress during resuscitated hemorrhagic shock in rats

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**Introduction**
Hydrogen sulfide (H2S) has been shown to improve survival in rodent models of lethal hemorrhage. Conversely, other authors have reported that inhibition of endogenous H2S production improves hemodynamics and reduces organ injury after hemorrhagic shock. Since all of these data originate from unresuscitated models and/or the use of a pre-treatment design, we therefore tested the hypothesis that the H2S donor, sodium hydrosulfide (NaHS), may improve hemodynamics in resuscitated hemorrhagic shock and attenuate oxidative and nitrosative stresses.

**Methods**
Thirty-two rats were mechanically ventilated and instrumented to measure mean arterial pressure (MAP) and carotid blood flow (CBF). Animals were bled during 60 minutes in order to maintain MAP at 40 ± 2 mm Hg. Ten minutes prior to retransfusion of shed blood, rats randomly received either an intravenous bolus of NaHS (0.2 mg/kg) or vehicle (0.9% NaCl). At the end of the experiment (T = 300 minutes), blood, aorta and heart were harvested for Western blot (inducible Nitric Oxide Synthase (iNOS), Nuclear factor-κB (NF-κB), phosphorylated Inhibitor κB (P-IκB), Inter-Cellular Adhesion Molecule (I-CAM), Heme oxygenase 1 (HO-1), Heme oxygenase 2 (HO-2), as well as nuclear respiratory factor 2 (Nrf2)). Nitric oxide (NO) and superoxide anion (O2 -) were also measured by electron paramagnetic resonance.

**Results**
At the end of the experiment, control rats exhibited a decrease in MAP which was attenuated by NaHS (65 ± 32 versus 101 ± 17 mmHg, P < 0.05). CBF was better maintained in NaHS-treated rats (1.9 ± 1.6 versus 4.4 ± 1.9 ml/minute P < 0.05). NaHS significantly limited shock-induced metabolic acidosis. NaHS also prevented iNOS expression and NO production in the heart and aorta while significantly reducing NF-κB, P-IκB and I-CAM in the aorta. Compared to the control group, NaHS significantly increased Nrf2, HO-1 and HO-2 and limited O2 - release in both aorta and heart (P < 0.05).

**Conclusions**
NaHS is protective against the effects of ischemia reperfusion induced by controlled hemorrhage in rats. NaHS also improves hemodynamics in the early resuscitation phase after hemorrhagic shock, most likely as a result of attenuated oxidative stress. The use of NaHS hence appears promising in limiting the consequences of ischemia reperfusion (IR).

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