



Cardiac and metabolic effects of hypothermia and inhaled hydrogen sulfide in anesthetized and ventilated mice

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Objective: To test the hypothesis whether inhaled hydrogen sulfide amplifies the effects of deliberate hypothermia during anesthesia and mechanical ventilation as hypothermia is used to provide organ protection after brain trauma or circulatory arrest. Awake mice inhaling hydrogen sulfide exhibit reduced energy expenditure, hypothermia, and bradycardia despite unchanged systolic heart function. In rodents, anesthesia alone causes decreased metabolic rate and thus hypothermia and bradycardia. Design: Prospective, controlled, randomized study. Setting: University animal research laboratory. Subjects: Male C57/B6 mice. Interventions: After surgical instrumentation (central venous, left ventricular pressure-conductance catheters, ultrasound flow probes on the portal vein and superior mesenteric artery), normo- or hypothermic animals (core temperature = 38°C and 27°C) received either 100 ppm hydrogen sulfide or vehicle over 5 hrs (3 hrs hydrogen sulfide during normothermia). Measurements and Main Results: During normothermia, hydrogen sulfide had no hemodynamic or metabolic effect. With or without hydrogen sulfide, hypothermia decreased blood pressure, heart rate, and cardiac output, whereas stroke volume, ejection fraction, and end-diastolic pressure remained unaffected. Myocardial and hepatic oxidative deoxyribonucleic acid damage (comet assay) and endogenous glucose production (rate of appearance of 1,2,3,4,5,6-13C6-glucose) were similar in all groups. Hypothermia comparably decreased CO₂ production with or without inhaled hydrogen sulfide. During hypothermia, inhaled hydrogen sulfide increased the glucose oxidation rate (derived from the expiratory 13CO₂/12CO₂ ratio). This shift toward preferential carbohydrate utilization coincided with a significantly attenuated responsiveness of hepatic mitochondrial respiration to stimulation with exogenous cytochrome-c-oxidase (high-resolution respirometry). Conclusions: In anesthetized and mechanically ventilated mice, inhaled hydrogen sulfide did not amplify the systemic hemodynamic and cardiac effects of hypothermia alone. The increased aerobic glucose oxidation together with the reduced responsiveness of cellular respiration to exogenous cytochrome-c stimulation suggest that, during hypothermia, inhaled hydrogen sulfide improved the yield of mitochondrial respiration, possibly via the maintenance of mitochondrial integrity. Hence, inhaled hydrogen sulfide may offer metabolic benefit during therapeutic hypothermia.

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