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Hydrogen is neuroprotective and preserves cerebrovascular reactivity in asphyxiated newborn pigs

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Abstract

Hydrogen (H2) has been reported to neutralize toxic reactive oxygen species. Oxidative stress is an important mechanism of neuronal damage after perinatal asphyxia. We examined whether 2.1% H2-supplemented room air (H2-RA) ventilation would preserve cerebrovascular reactivity (CR) and brain morphology after asphyxia/reventilation (A/R) in newborn pigs. Anesthetized, ventilated piglets were assigned to one of the following groups: A/R with RA or H2-RA ventilation (A/R-RA and A/R-H2-RA; n = 8 and 7, respectively) and respective time control groups (n = 9 and 7). Asphyxia was induced by suspending ventilation for 10 min, followed by reventilation with the respective gases for 4 h. After euthanasia, the brains were processed for neuropathological examination. Pial arteriolar diameter changes to graded hypercapnia (5-10% CO2 inhalation), and NMDA (10(-4) M) were determined using the closed cranial window/intravital microscopy before and 1 h after asphyxia. Neuropathology revealed that H2-RA ventilation significantly reduced neuronal injury induced by A/R in virtually all examined brain regions including the cerebral cortex, the hippocampus, basal ganglia, cerebellum, and the brainstem. Furthermore, H2-RA ventilation significantly increased CR to hypercapnia after A/R (% vasodilation was 23 ± 4% versus 41 ± 9%, $p < 0.05$). H2-RA ventilation did not affect reactive oxygen species-dependent CR to NMDA. In summary, H2-RA could be a promising approach to reduce the neurologic deficits after perinatal asphyxia.

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