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Delayed neurovascular dysfunction is alleviated by hydrogen in asphyxiated newborn pigs

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Abstract

Background: The neurovascular unit encompasses the functional interactions of cerebrovascular and brain parenchymal cells necessary for the metabolic homeostasis of neurons. Previous studies indicated marked but only transient (1-4 h) reactive oxygen species-dependent neurovascular dysfunction in newborn pigs after severe hypoxic/ischemic (H/I) stress contributing to the neuronal injury after birth asphyxia.

Objectives: Our major purpose was to determine if neurovascular dysfunction would also occur later, at 24 h after a milder H/I stress. We also tested if the putative hydroxyl radical scavenger hydrogen (H2) exerted neurovascular protection.

Methods: Anesthetized, ventilated piglets were assigned to three groups of 9 animals: time control, asphyxia/reventilation with air, and asphyxia/reventilation with air +2.1% H2 for 4 h. Asphyxia was induced by suspending ventilation for 8 min. Cerebrovascular reactivity (CR) of pial arterioles was determined using closed cranial window/intravital microscopy 24 h after asphyxia to the endothelium-dependent cerebrovascular stimulus hypercapnia, the neuronal function-dependent stimulus N-methyl-D-aspartate (NMDA), norepinephrine, and sodium nitroprusside. The brains were subjected to histopathology.

Results: Hemodynamic parameters, blood gases, and core temperature did not differ significantly among the experimental groups. In the early reventilation period, the recovery of electroencephalographic activity was significantly better in H2-treated animals. Asphyxia/reventilation severely attenuated CR to hypercapnia and NMDA; however, reactivity to norepinephrine and sodium nitroprusside were unaltered. H2 fully or partially preserved CR to hypercapnia or NMDA, respectively. Histopathology revealed modest neuroprotection afforded by H2.

Conclusions: Severe stimulus-selective delayed neurovascular dysfunction develops and persists even after mild H/I stress. H2 alleviates this delayed neurovascular dysfunction that can contribute to its neuroprotective effect.

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