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Coadministration of hydrogen gas as part of the carrier gas mixture suppresses neuronal apoptosis and subsequent behavioral deficits caused by neonatal exposure to sevoflurane in mice

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Abstract

Background: In animal models, several anesthetics induce widespread increases in neuronal apoptosis in the developing brain with subsequent neurologic deficits. Although the mechanisms are largely unknown, the neurotoxicity may, at least in part, be due to elevated oxidative stress caused by mitochondrial dysfunction. In an investigation of potential therapies that could protect against this type of damage, we studied the effects of molecular hydrogen on anesthetic-induced neurotoxicity in the developing mouse brain.

Methods: Six-day-old C57BL/6 mice were exposed to 3% sevoflurane for 6 h with or without hydrogen (< 1.3%) as part of the carrier gas mixture. Apoptosis was evaluated by immunohistochemical staining for cleaved caspase-3 (n = 8-10/group). Western blot analysis for cleaved poly-(adenosine diphosphate-ribose) polymerase was also performed to examine apoptosis (n = 3-6/group). Oxidative stress was assessed by immunohistochemical staining for 4-hydroxy-2-nonenal (n = 8/group). Long-term memory and social behavior were examined using the fear conditioning test and the sociability test, respectively (n = 18-20/group).

Results: Western blot analysis showed that coadministration of 1.3% hydrogen gas significantly ($P < 0.001$) reduced the level of neuronal apoptosis to approximately 40% compared with sevoflurane exposure alone. Immunohistochemical analysis showed that hydrogen reduced oxidative stress induced by neonatal sevoflurane exposure. Although neonatal sevoflurane exposure caused impairment in long-term memory and abnormal social behaviors in adulthood, mice coadministered hydrogen gas with sevoflurane did not exhibit these deficits.

Conclusions: Inhalation of hydrogen gas robustly decreased neuronal apoptosis and subsequent cognitive impairments caused by neonatal exposure to sevoflurane.

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