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Hydrogen gas attenuates sevoflurane neurotoxicity through inhibiting nuclear factor κ-light-chain-enhancer of activated B cells signaling and proinflammatory cytokine release in neonatal rats

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Abstract

Anesthesia neurotoxicity in developing brain has gained increasing attention. However, knowledge regarding its mitigating strategies remains scant. Sevoflurane, a commonly used anesthetic, is responsible for learning and memory deficits in neonates. Molecular hydrogen is reported to be a potential neuroprotective agent because of its antioxidative and anti-inflammatory activities. This study aimed to investigate the effect of hydrogen gas on sevoflurane neurotoxicity. The newborn rats were treated with sevoflurane and/or hydrogen gas for 2 h. Spatial recognition memory and fear memory were determined by Y-maze and fear conditioning tests at 10 weeks of age. Nuclear factor κ-light-chain-enhancer of activated B cells (NF-κB) and proinflammatory cytokine levels were detected using western blot analysis. The data showed that the spatial recognition memory and fear memory of the rats treated with sevoflurane decreased compared with the control, and the cognitive function of the rats treated with sevoflurane and hydrogen gas significantly increased in comparison with treatment with sevoflurane alone. Moreover, hydrogen gas suppressed NF-κB phosphorylation and nuclear translocation and reduced the production of interleukin-1β, interleukin-6, and tumor necrosis factor-α following sevoflurane administration. Thus, the results proposed that hydrogen gas might protect against sevoflurane neurotoxicity by inhibiting NF-κB activation and proinflammatory cytokine release, providing a novel therapeutic strategy for anesthesia neurotoxicity.

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