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Hydrogen supplemented air inhalation reduces changes of prooxidant enzyme and gap junction protein levels after transient global cerebral ischemia in the rat hippocampus

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Abstract

Transient global cerebral ischemia (TGCI) occurs during acute severe hypotension depriving the brain of oxygen and glucose for a short period of time. During reperfusion, several mechanisms can induce secondary neuronal damage, including the increased production of reactive oxygen species (ROS). Hydrogen gas-enriched air inhalation is a neuroprotective approach with proven antioxidant potential, which has not yet been examined in TGCI. Accordingly, we set out to describe the effect of inhalation of 2.1% hydrogen supplemented room air (H(2)-RA) in comparison with a well studied neuroprotective agent, rosiglitazone (RSG) in a TGCI rat model. Male Wistar rats were exposed to TGCI (n=26) or sham operation (n=26), while a third group served as intact control (naive, n=5). The operated groups were further divided into non-treated, H(2)-RA, RSG (6 mg/kg i.v.) and vehicle treated animals. Tissue samples from the hippocampus and frontal cortex were taken 3 days following surgery. Western blot analysis was applied to determine the expressions of cyclooxygenase-2 (COX-2), neuronal and endothelial nitric oxide synthase (nNOS and eNOS, respectively), manganese superoxide dismutase (MnSOD) and glial connexin proteins: connexin 30 and connexin 43. The expressions of COX-2, and connexin proteins were upregulated, while nNOS was downregulated 3 days after TGCI. Both RSG and H(2)-RA prevented the changes of enzyme and connexin levels. Considering the lack of harmful side effects, inhalation of H(2)-RA can be a promising approach to reduce neuronal damage after TGCI.

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