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Hydrogen inhalation is neuroprotective and improves functional outcomes in mice after intracerebral hemorrhage

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Abstract

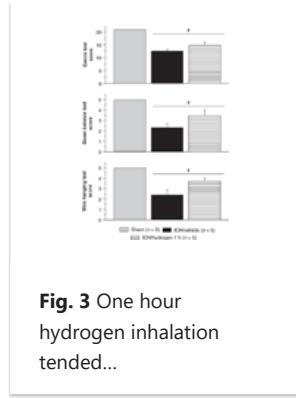
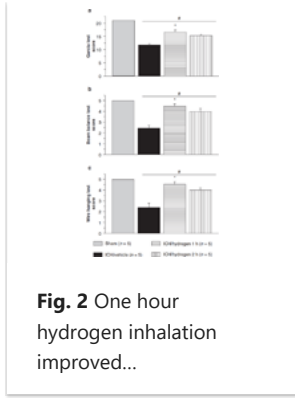
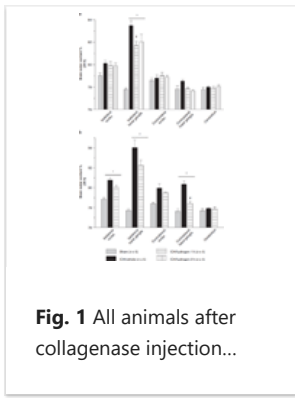
Objective: Oxidative stress contributes significantly to the development of secondary brain injury after intracerebral hemorrhage (ICH). It has been previously demonstrated that hydrogen gas can decrease oxidative stress by scavenging reactive oxygen species. We hypothesized that hydrogen therapy will reduce brain oxidative stress in mice after ICH and thereby will lead to reduced brain edema and improved neurological outcomes.

Materials and methods: CD1 male mice (weight 30-35 g) were divided into the following groups: sham, ICH+vehicle (room air), ICH+1-h hydrogen treatment, and ICH+2-h hydrogen treatment. ICH was induced by injection of bacterial collagenase into the right basal ganglia. The evaluation of outcomes was done at two time points: 24 and 72 h post-ICH. Brain water content was measured for assessment of brain edema (wet/dry weight method), and three neurological tests were performed pre- and postoperatively.

Results: Collagenase injection was found to induce brain edema and impair functional performance of rats. The hydrogen inhalation reduced these effects acutely (24 h); however it exhibited only a tendency to improvement in the delayed study (72 h).

Conclusions: Our results suggest that hydrogen inhalation exerts an acute brain-protective effect in the mouse ICH model. However, the acute hydrogen therapy alone is not sufficient to improve delayed ICH outcomes in this model.

Figures



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