The long-term effect of sevoflurane on neuronal cell damage and expression of apoptotic factors after cerebral ischemia and reperfusion in rats.

We investigated the long-term effects of sevoflurane on histopathologic injury and key proteins of apoptosis in a rat hemispheric ischemia/reperfusion model. Sixty-four male Sprague-Dawley rats were randomly assigned to Group 1 (fentanyl and N₂O/O₂; control) and Group 2 (2.0 vol% sevoflurane and O₂/air). Ischemia (45 min) was produced by unilateral common carotid artery occlusion plus hemorrhagic hypotension (mean arterial blood pressure 40 mm Hg). Animals were killed after 1, 3, 7, and 28 days. In hematoxylin and eosin-stained brain sections eosinophilic hippocampal neurons were counted. Activated caspase-3 and the apoptosis-regulating proteins Bax, Bcl-2, Mdm-2, and p53 were analyzed by immunostaining. No eosinophilic neurons were detected in sevoflurane-anesthetized rats over time, whereas 9%-38% of the hippocampal neurons were eosinophilic (days 1-28) in control animals. On days 1 and 3, the concentration of Bax was 140%-200% larger in fentanyl/N₂O-anesthetized animals compared with sevoflurane. Bcl-2 was 100% less in control animals during the first 3 days. Activated caspase-3 was detected in neurons of both groups (0.75%-2.2%). These data support a sustained neuroprotective potency of sevoflurane related to reduced eosinophilic injury after cerebral
ischemia/reperfusion.

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