Sevoflurane affects neurogenesis after forebrain ischemia in rats.

BACKGROUND: The effect of sevoflurane on the neuroregenerative potential after neuronal injury is unclear. We investigated the effect of low and high concentrations of sevoflurane on endogenous neurogenesis after cerebral ischemia.

METHODS: Anesthetized and ventilated rats were randomized to four different treatment groups. Groups 1 and 2: 1.4% sevoflurane; Groups 3 and 4: 2.8% sevoflurane. In Groups 1 and 3, no cerebral ischemia was induced (sham-operated). In Groups 2 and 4, 10 min of forebrain ischemia was induced by bilateral carotid artery occlusion plus hemorrhagic hypotension. Physiological variables were maintained constant. Bromodeoxyuridine was given as a marker of neurogenesis. After 28 days brains were perfused.

Histopathological damage of the hippocampus was evaluated in hematoxylin and eosin (HE) stained sections using the HE-index (0 = no damage; 1 = 1%-10% damage; 2 = 11%-50% damage; 3 = 51%-100% damage). Immunohistochemistry was used to detect bromodeoxyuridine-positive neurons. Eight untreated rats were investigated as naive controls (Group 5).

RESULTS: In neither sham-operated group was histopathological damage or change in neurogenesis observed compared to naive controls. In rats anesthetized with 1.4% sevoflurane, cerebral ischemia caused mild
neuronal damage (HE-index of 0.64 +/- 0.84) and increased neurogenesis by 60% when compared with respective sham-operated animals; with 2.8% sevoflurane, the HE-index was 1.22 +/- 1.14, and the number of newly generated neurons increased by 230% when compared with respective sham-operated animals. CONCLUSION: The present data suggest that high concentrations of sevoflurane stimulate neurogenesis in the dentate gyrus after cerebral ischemia.