Sevoflurane anesthesia improves cognitive performance in mice, but does not influence in vitro long-term potentiation in hippocampus CA1 stratum radiatum.

Abstract:
Whether the occurrence of postoperative cognitive dysfunction is a result of the effects of surgery or anesthesia is under debate. In this study, we investigated the impact of sevoflurane anesthesia on cognitive performance and cellular mechanisms involved in learning and memory. Male C57Bl6/J mice (4-5 months) were exposed to one minimum alveolar concentration sevoflurane for two hours. After 24 h, cognitive performance of mice was assessed using the modified hole board test. Additionally, we evaluated hippocampal long-term potentiation and expression levels of different receptor subunits by recording excitatory postsynaptic field potentials and using the western blot technique, respectively. Non-anesthetized mice served as controls. In anesthetized mice, neither cognitive performance nor long-term potentiation was impaired 24 h after anesthesia. Interestingly, sevoflurane anesthesia induced even an improvement of cognitive performance and an elevation of the expression levels of N-methyl-D-aspartate (NMDA) receptor type 1 and 2B subunits in the hippocampus. Since NMDA receptor type 1 and 2B subunits play a crucial role in processes related to learning and memory, we hypothesize that sevoflurane-induced changes in
NMDA receptor subunit composition might cause hippocampus-dependent cognitive improvement. The data of the present study are in favor of a minor role of anesthesia in mediating postoperative cognitive dysfunction.