Dokumenttyp: journal article

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Titel des Beitrags:
Isoflurane anaesthesia reversibly improves cognitive function and long-term potentiation (LTP) via an up-regulation in NMDA receptor 2B subunit expression.

Abstract:
Postoperative cognitive dysfunction (POCD) is a decline in cognitive performance after a surgery performed under anaesthesia. The exact roles of surgery and/or anaesthesia for facilitating POCD are unclear. This study investigates the effects of isoflurane anaesthesia on cognitive performance and cellular mechanisms involved in learning and memory function. Male C57BL6/J mice (age: 4-5 months) were anaesthetized with isoflurane in oxygen/air (FiO(2)=0.5) for 2h, non-anaesthetized mice served as controls. After 24h, neurocognitive function, in vitro long-term potentiation (LTP), or protein expression were evaluated. In a visuospatial test, anaesthetized mice showed better cognitive performance as they learned faster compared to controls. In hippocampal slices of anaesthetized mice, in vitro LTP was enhanced as reflected in an increased extracellular field potential (fEPSP) slope after 1h to 210.2+/-17% (control: 156.8+/-7.2%; n=14; p<0.05). NR2B subunits of the NMDA receptors were selectively up-regulated in hippocampal neurones after anaesthesia. Blocking these receptors either with the NR2B selective antagonists ifenprodil or RO25-6981 (R-(R,S)-alpha-(4-hydroxyphenyl)-beta-methyl-4-(phenylmethyl)-1-piperidine propranol), prevents the
anaesthesia-induced improvement in cognitive function as well as enhancement of in vitro LTP. The anaesthesia-mediated effects on NR2B subunits were fully reversed to control levels seven days after anaesthesia. The present data suggests that isoflurane anaesthesia induces a hippocampus-specific elevation of NR2B subunit composition, enhances LTP in CA1 neurones, and produces hippocampal-dependent cognitive improvement.

Zeitschriftentitel / Abkürzung:
Neuropharmacology

Jahr: 2009

Band: 56

Heft / Issue: 3

Seiten: 626-36

Sprache: eng


Print-ISSN: 0028-3908

TUM Einrichtung: sthesiologie

Occurences:
- Einrichtungen > Fakultäten > Fakultät für Medizin > Kliniken und Institute > Klinik für Anästhesiologie > Klinik für Anästhesiologie (DHM) > 2009

entries: