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Titel des Beitrags:
Xenon attenuates excitatory synaptic transmission in the rodent prefrontal cortex and spinal cord dorsal horn.

Abstract:
BACKGROUND: The molecular mechanisms of the inhalational anesthetic xenon are not yet fully understood. Recently, the authors showed that xenon reduces both N-methyl-d-aspartate (NMDA) and alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor-mediated synaptic transmission in a brain slice preparation of the amygdala. In the current study, the authors examined the effects of xenon on synaptic transmission in the prefrontal cortex and the spinal cord dorsal horn (substantia gelatinosa). METHODS: In rodent brain or spinal cord slice preparations, the authors used patch clamp technique to investigate the impact of xenon on NMDA and AMPA receptor-mediated excitatory postsynaptic currents, as well as on gamma-aminobutyric acid type A receptor-mediated inhibitory postsynaptic currents. The currents were either evoked upon electrical stimulation (NMDA-eEPSCs and AMPA-eEPSCs) or upon photolysis of caged L-glutamate (p-NMDA-Cs and p-AMPA-Cs). In addition, the authors investigated the effects of xenon on AMPA receptor-mediated miniature excitatory postsynaptic currents. RESULTS: In both central nervous system regions, xenon had virtually no effect on inhibitory postsynaptic currents. In the prefrontal cortex (spinal cord), xenon reversibly reduced NMDA-eEPSCs to approximately 58% (72%) and AMPA-eEPSCs to approximately 67%
(65%) of control. There was no difference in the xenon-induced reduction of NMDA-eEPSCs and p-NMDA-Cs, or AMPA-eEPSCs and p-AMPA-Cs. Xenon did not affect the frequency of miniature excitatory postsynaptic currents but reduced their amplitude. CONCLUSIONS: In the current study, the authors found that xenon depresses NMDA and AMPA receptor-mediated synaptic transmission in the prefrontal cortex and the substantia gelatinosa without affecting gamma-aminobutyric acid type A receptor-mediated synaptic transmission. These results provide evidence that the effects of xenon are primarily due to postsynaptic mechanisms.