Isoflurane modulates glutamatergic and GABAergic neurotransmission in the amygdala.

Attempts have been made to attribute the particular features of general anaesthesia such as hypnosis, analgesia, amnesia and autonomic stability to certain brain regions. In the present study, we examined the effects of the commonplace volatile anaesthetic isoflurane on synaptic transmission in an in vitro slice preparation of the murine amygdala. Despite the established role of this limbic structure in the formation of aversive memories, conditioned fear and anxiety, as well as pain processing and regulation of sympathetic tone, the influence of volatile anaesthetics on synaptic signalling has not yet been investigated in this region of the brain.

Evoked postsynaptic currents were monitored from principal neurons in the basolateral nucleus of the amygdala by means of patch-clamp recording. The mixed postsynaptic currents were mediated by non-NMDA, NMDA, GABA A and GABA B receptors. Isoflurane added to the perfusion medium reduced the strength of synaptic signalling following the activation of non-NMDA, NMDA, and GABA B receptors, whereas the GABA A receptor-mediated responses were enhanced. The overall reduction of neuronal excitability was also reflected in a reduction of field potential amplitudes. Isoflurane neither changed the membrane resting potential nor the input resistance of
principal neurons in the amygdala. The present results may contribute to the understanding of how stress reactions and long-lasting neuroplastic processes are suppressed under general anaesthesia.