Sevoflurane impairs cerebral blood flow autoregulation in rats: reversal by nonselective nitric oxide synthase inhibition.

Abstract: In this study, we investigated the effects of 1.0 and 2.0 minimum alveolar anesthetic concentration (MAC) sevoflurane on cerebral blood flow (CBF) autoregulation before and after nonselective inhibition of nitric oxide (NO) synthase in rats. Rats were randomly assigned as follows: Group 1 (n = 8): 1.0 MAC sevoflurane; Groups 2 and 3 (n = 8 per group): 2.0 MAC sevoflurane. Assessment of autoregulation within a mean arterial blood pressure range of 140-60 mm Hg was performed by graded hemorrhage before and after administration of l-arginine methyl ester (l-NAME, 30 mg/kg IV, Groups 1 and 2) or during hypocapnia (Group 3). In 10 additional animals, brain tissue NO\textsubscript{2}(\text{-}) concentrations were measured at 1.0 and 2.0 MAC sevoflurane. CBF autoregulation was maintained with 1.0 MAC sevoflurane (Group 1) regardless of NO synthase status indicating that CBF autoregulation might not be related to NO availability. Sevoflurane dose-dependently increased brain tissue NO\textsubscript{2}(\text{-}) and impaired CBF autoregulation. Administration of l-NAME (Group 2) but not hypocapnia (Group 3) restored CBF autoregulation. This suggests that sevoflurane impairs the autoregulatory capacity secondary to an increase of the perivascular NO availability and questions the importance of basal cerebrovascular tone in terms of vasodilatory capacity during hypotensive challenges.
IMPLICATIONS: The present study suggests that the volatile anesthetic sevoflurane dose-dependently impairs cerebrovascular autoregulation by mechanisms secondary to increase of perivascular nitric oxide availability.