ABSTRACT

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THE EFFECTS OF SEVOFLURANE, PROPOFOL AND NITROUS OXIDE ON REGIONAL CEREBRAL BLOOD FLOW, OXYGEN CONSUMPTION AND BLOOD VOLUME

Positron Emission Tomography and EEG Studies on Healthy Subjects

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Anesthetic drugs have profound effects on cerebral electric activity, metabolism and perfusion. Intravenous anesthetics like propofol can reduce neuronal activity even down to the level of electroencephalographic suppression, simultaneously almost halving the rate of cerebral metabolism, i.e. the consumption of oxygen and glucose. Due to so-called 'flow-activity coupling', also regional cerebral blood flow reduces accordingly. For volatile anesthetics like sevoflurane, the data are contradictory. Although metabolism is effectively reduced, global cerebral blood flow is either increased or decreased in various studies. Results on adjunct nitrous oxide also vary, showing mostly increased metabolism and perfusion. The purpose of the present study was, by using positron emission tomography, to quantitatively compare the effects of propofol and sevoflurane alone and with nitrous oxide on cerebral perfusion and metabolism at different electroencephalographically verified surgical levels of pseudo-steady-state anesthesia.

Both sevoflurane and especially propofol reduced quantitative blood flow. The effects were maintained at increasing concentrations of propofol (2 EC_{50}; Effective concentration 50), whereas sevoflurane caused a redistribution of flow at highest dose level (2 MAC; Minimal alveolar concentration). Adjunct nitrous oxide increased cerebral blood flow with either anesthetic, especially abolishing the flow-reducing effect of sevoflurane. Both drugs reduced relative flow especially in the thalamus, the cuneus and the fronto-parietal cortex, sevoflurane additionally in the cerebellum, and propofol in the anterior cingulate. During propofol the oxygen extraction fraction was maintained at the awake level, even with nitrous oxide. Sevoflurane, but especially the combination of sevoflurane and nitrous oxide caused luxury perfusion (in the whole cortex and cerebellum). Indirectly this supports avoiding the use of nitrous oxide or high dose sevoflurane in patients with space occupying intra-cranial processes.

Both sevoflurane and propofol reduced electroencephalographic activity dose dependently until burst suppression. The electroencephalographic monitoring gave unexpected evidence that sevoflurane, but not propofol, was dose-dependently epileptogenic. Different epileptiform complexes appeared in fixed order, and in 3 out of 8 subjects secondarily generalized partial epileptiform discharges were recorded at deepest level of sevoflurane anesthesia. This supports the use of antiepileptic drugs if prolonged seizure is suspected during sevoflurane anesthesia. The clinical relevance of epileptiform patterns needs urgently further investigations.