

Anesthetic Effects on Cerebral Blood Flow

CoreNotes by Core Concepts Anesthesia Review, LLC

What You Must Know

1. There exists Flow-Metabolism Coupling in the CNS such that changes in cerebral oxygen consumption ($CMRO_2$) result in concomitant changes in cerebral blood flow (CBF).
2. Propofol, etomidate, benzodiazepines and barbiturates all reduce $CMRO_2$, CBF and intracranial pressure (ICP).
3. Ketamine increases $CMRO_2$, CBF and ICP.
4. Volatile anesthetics decrease $CMRO_2$. However, the direct vasodilatory effect of the volatile agents causes an increase in CBF and ICP.
5. N_2O increases CBF and ICP, but has little effect on $CMRO_2$.

$CMRO_2$ and CBF are tightly linked. Increases in cerebral metabolic demand result in an increase in CBF. Conversely, decreases in cerebral metabolic demand result in a decrease in CBF. This process is known as flow-metabolism coupling. Since the intracranial contents consist of brain, CSF and blood volume, an increase in CBF is associated with a rise in ICP.

Drugs that reduce $CMRO_2$ cause a decrease in CBF and ICP. These drugs include all of the intravenous induction agents (except ketamine), benzodiazepines and narcotics. Ketamine causes an increase in $CMRO_2$, CBF and ICP. For this reason, ketamine has limited usefulness in neuroanesthesia.

Although the volatile anesthetic agents depress $CMRO_2$, their direct vasodilatory effect causes a loss of flow-metabolism coupling. As a result, the volatile anesthetic agents cause an increase in both CBF and ICP despite causing a decrease in $CMRO_2$.

Nitrous oxide is a potent cerebral vasodilator and produces an increase in both CBF and ICP. The combination of volatile anesthetic agent with nitrous oxide may as much as triple CBF. Many neuroanesthesia providers advocate the avoidance of nitrous oxide in neurosurgical patients.

Additional Reading:

Nagelhout, JJ, and Plaus, KL. *Nurse Anesthesia*. St. Louis: Elsevier, 2013:700-703