



Clinical visualization of cerebral vasodilatation by desflurane

Mathangi Krishnakumar, MD, PDF · Venkatapura Ramesh, MD · Amit Goyal, DNB ·
Nupur Pruthi, MCh

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The effect of volatile anesthetics on cerebral blood flow (CBF) depends on the balance between the agent's direct vasodilatory action and the indirect vasoconstrictive action mediated by flow-metabolism coupling. Transcranial Doppler measurement and CBF assessment using argon gas have shown a vasodilatory effect of desflurane at more than 1.5 minimum alveolar concentration (MAC).^{1,2} But no experimental studies on human cerebral vessels have shown any change.

Here, we present a case in which vasospasm induced by vessel manipulation in a patient with moyamoya disease was apparently reversed by increasing desflurane concentration to the MAC (> 1).

An eight-year-old female with moyamoya disease underwent a left superficial temporal artery to left middle cerebral artery bypass under general anesthesia. The patient was induced with fentanyl $2 \mu\text{g}\cdot\text{kg}^{-1}$ and thiopentone $5 \text{ mg}\cdot\text{kg}^{-1}$ followed by intubation after paralysis with atracurium $0.5 \text{ mg}\cdot\text{kg}^{-1}$. The anesthesia was maintained with air and oxygen mixture with desflurane at a MAC between 0.8 and 1 titrated to a spectral entropy value of 40–50. After isolation of the middle cerebral artery, vasospasm of the vessel was noticed (Figure A). The MAC of desflurane was increased from 0.8 to 1.3 over five minutes and vasodilation of the vessel was observed (Figure B). During this period, the blood pressure and end-

M. Krishnakumar, MD, PDF (✉) · V. Ramesh, MD ·
A. Goyal, DNB
Department of Neuroanaesthesia and Neurocritical care,
National Institute of Mental Health and Neurosciences,
Bengaluru, Karnataka, India
e-mail: mathz89@gmail.com

N. Pruthi, MCh
Department of Neurosurgery, National Institute of Mental Health
and Neurosciences, Bengaluru, Karnataka, India

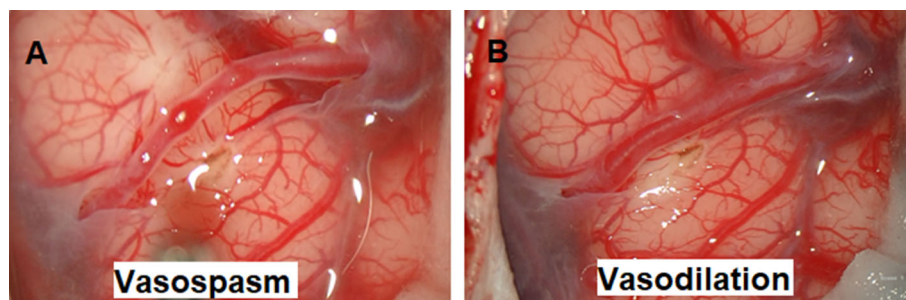


Figure Vasospasm in the middle cerebral artery of an 8-year-old female patient with moyamoya disease undergoing left superficial temporal artery to left middle cerebral artery (MCA) bypass under

general anesthesia. At a minimum alveolar concentration (MAC) of 0.8 desflurane vasospasm (A) can be seen in the MCA, which resolves when the desflurane is increased to 1.3 MAC (B)

tidal carbon dioxide were maintained constant and no other vasodilators (e.g., papaverine) were used.

Conflicts of interest None.

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