KJA
Korean Journal of Anesthesiology

Editorial
pISSN 2005-6419 • eISSN 2005-7563

Can desflurane be an alternative to sevoflurane in neuroanesthesia?

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In neuroanesthesia, the mechanism of impact of the anesthetics and techniques on the cerebral blood flow (CBF) is important. Similar to intravenous sedative-hypnotics, inhalational anesthetics, with the exception of halothane, suppress the cerebral metabolic rate (CMR) and can produce burst-suppression on electroencephalogram. Dose-dependent effects of inhalational anesthetics on CBF can be caused by the direct effects on the vascular smooth muscles. With doses lower than 1 minimal alveolar concentration (MAC), CBF is modestly decreased. With doses higher than 1 MAC, direct cerebral vasodilation results in an increase in CBF and cerebral blood volume. Therefore, the net effect of inhalational anesthetics on CBF is a balance between CBF reduction due to CMR suppression and CBF elevation due to direct cerebral vasodilation.

There are two mechanisms control CBF: autoregulation, which sustains the flow when the perfusion pressure changes, and brain metabolism, which adjusts the flow to meet the metabolic needs. A major component of the latter is the reactivity of the cerebral blood vessels to CO_2 and O_2. CO_2 increases the CBF and arterial blood pressure. CBF increases not only from the effect of CO_2 vasodilatation but also from the increased pressure of perfusion after the autoregulation is exhausted. In neuroanesthesia, $PaCO_2$ is reduced to decrease CBF and to thereby decrease the intracranial pressure. A change in cerebrovascular resistance or CBF in response to changes in $PaCO_2$ is termed cerebrovascular CO_2 reactivity (CCO_2R). In neuroanesthesia, the effects of various anesthetics on CCO_2R may be important.

In a previous systematic review on the CCO_2R under anesthesia, CCO_2R was maintained with both inhalational and intravenous agents within the anesthetic concentrations used clinically [1], but the degree of reactivity and responses to the hypercapnic and hypocapnic ranges of CO_2 were different among individual agents. However, most of the available information had been collected from non-neurosurgical patients, and the studies had the limitation of significant methodological heterogeneity.

Desflurane acts as a positive and negative allosteric modulator of γ-aminobutyric acid ($GABA_A$) and glycine receptors and of the nicotinic acetylcholine receptor, respectively, and affects other ligand-gated ion channels. It has the most rapid onset and offset among the volatile anesthetic drugs used for general anesthesia because of its low solubility in blood, which can be an advantage in neurosurgery, as it enables neurological examination to be performed soon after the surgery. The vasodilatory effects of desflurane and isoflurane are greater than those of sevoflurane in normocapnia. Previous studies have shown the effect of desflurane not only on cerebrovasculature under hypercapnia but also on intracranial pressure inconsistently.

As published in the current issue of the *Korean Journal of Anesthesiology*, Dr. Sakata and his colleagues [2] confirmed that desflurane induces cerebrovascular responses similar to those of sevoflurane in a rat model. They concluded that desflurane can be used as safely as sevoflurane in neuroanesthesia. Although some readers may question the involvement of the pia mater vessels, which might not be the target vessels for clinical practitioners, responses in these vessels can be directly observed in vivo using the closed cranial window preparation, along with typical cerebrovascular reactions. In this study, the author administered 1.0 MAC of volatile anesthetic during hypercapnia, which might make the readers curious regarding the direct observation of vessel changes with 0.5 MAC of volatile anesthetics during hypercapnia. Because the responses of the pia mater ves-

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Received: May 9, 2019.
Accepted: May 9, 2019.

Korean J Anesthesiol 2019 June 72(3): 207-208
https://doi.org/10.4097/kja.19199

sels at 1.0 MAC were larger than those at 0.5 MAC, the authors seemed to think that 1.0 MAC would be sufficient to observe vasodilatory responses induced by inhalational anesthetics under hypercapnia. However, the possibility that the pial vessel reactivity observed in this study might be affected by the presence of pentobarbital needs to be explored in future studies [3].

The authors investigated the effects of desflurane on the cerebral vascular (pial vessel) responses induced by hypercapnia in rats and compared the effects of desflurane and sevoflurane, which have been directly observed in a few experiments on cerebral vessels. However, there is still little clinical evidence to support the author's arguments. Therefore, a definite conclusion cannot be drawn, and further studies need to be conducted. If the results of future studies on humans support the results of this study, then this study can be used a reference for the merits of desflurane in neuroanesthesia.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

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