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Re: Nakamura. Implications of the suppression of guanylate cyclase activity by halothane

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In reply: Although I do not know how much of sodium nitroprusside (SNP) produced how much NO, volatile anesthetics (VA) failed to inhibit NO-stimulated soluble guanylyl cyclase (sGC) activity at high NO concentrations in our incubations [1]. If SNP at the concentration (even if it is not a maximum dose) used in the study of Terasako et al. [2] produced a large amount of NO, halothane and isoflurane at single concentration would not decrease cGMP formation, as they Stated in the discussion of their paper. To examine the effects of VA on sGC, the effects of VA at several concentrations on sGC which is stimulated by the wide range of SNP concentrations is to be determined. As I replied in the last letter [3], the different results may arise from the different experimental conditions (including the differences between the brain slice experiment and that with a soluble fraction). The effects of VA on sGC stimulated by SNP and NO solution might differ in brain slice and soluble fraction, respectively. Some site and mechanism between the glutamate receptor and NO formation may be more susceptible to halothane than sGC activity itself. However, halothane and sevoflurane clearly inhibited the NO-stimulated sGC activity in rat brain soluble fraction in our experimental conditions.

References

1. Masaki E, Kondo I (1998) Attenuation of nitric oxide-stimulated soluble guanylyl cyclase from the rat brain by halogenated volatile anesthetics. J Anesth 12:81–86
2. Terasako K, Nakamura K, Miyawaki I, Toda H, Kakuyama M, Mori K (1994) Inhibitory effects of anesthetics on cyclic guano-sine monophosphate (cGMP) accumulation in rat cerebellar slices. Anesth Analg 79:91–96
3. Masaki E (1999) Re: Teresako. Does halothane or sevoflurane inhibit NO-stimulated soluble guanylate cyclase activity under physiological conditions? J Anesth 13:62

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