

CORRESPONDENCE

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Editorial Note:—Dr. Shulman and Ms. Robillard provide a useful and easily accomplished suggestion to reduce the frequency of bibliographic citation errors in papers published in *ANESTHESIOLOGY*. Accordingly, we shall include a version of the above quoted paragraph as a part of each letter to authors of papers for which a revision is requested.

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Editor in Chief

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Halothane and the Guanylate Cyclase System

To the Editor:—Eskinder *et al.*¹ recently demonstrated that halothane stimulates particulate guanylate cyclase activity and increases tissue cyclic guanosine 3,5-monophosphate (cGMP) level in vascular smooth muscle of canine cerebral artery. It is a valuable contribution to the understanding of the mechanisms that underlie cerebral vasodilator effect of halothane. However, their description that the direct effect of halothane on vascular smooth muscle tissue cGMP levels had not been reported before their study is not correct.

We have previously reported that halothane increases cGMP content of endothelium-denuded aorta of the rat.² However, cGMP content of aorta with intact endothelium was rather decreased by halothane.³ A possible explanation for these observations is that halothane decreased basal production of endothelium-derived relaxing factor (EDRF), and thereby decreased endothelium-dependent cGMP formation, while increasing endothelium-independent cGMP. These results with rat aorta suggested that the stimulating effect of halothane on guanylate cyclase does not play a significant role in the vasodilator effect of halothane in physiologic conditions.

It is a common observation that cerebral artery is more susceptible to the dilator effect of halothane than are extracerebral arteries such as rat aorta. The finding of Eskinder *et al.* that cGMP level of cerebral artery with intact endothelium was increased by halothane, in contrast to our finding in rat aorta, is consistent with the high susceptibility of cerebral artery to halothane's dilator effect. However, their finding does not necessarily support the hypotheses that stimulating effect of halothane on guanylate cyclase differs in cerebral and extracerebral arteries, because we cannot deny the possibility that the effect of halothane on endothelium (*i.e.*, basal level of EDRF or potential of halothane to reduce it) differs in cerebral and extracerebral arteries.

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