Antagonism of Histamine-Induced Bronchoconstriction

To the Editor—Tobias et al. suggest that halothane prevents the bronchodilator effect of aminophylline by preventing the release of endogenous catecholamines. In support of this claim is: 1) the observation that aminophylline does not reverse a histamine-induced increase in airway resistance in the presence of halothane (1.5 MAC); and 2) that the addition of propranolol prevents the action of aminophylline.

We have previously shown that in humans half this concentration of halothane (0.8 MAC) has as powerful a bronchodilating effect as that of albuterol. Furthermore, when histamine is administered to humans following pretreatment with either albuterol or aminophylline, the former completely blocks the bronchoconstriction, whereas the latter only partially blocks the effect. Thus in conscious humans, aminophylline at maximal clinical dose is a weak antagonist of histamine-induced bronchoconstriction. Tobias et al. clearly show that there is no added effect of aminophylline in the presence of 1.5 MAC halothane and argue that this is because halothane blocks the release of catecholamine by aminophylline. However, I would argue that 1.5 MAC of halothane is exerting a maximum bronchodilator effect that cannot be exceeded by adding aminophylline. It remains to be demonstrated that the increase in airway resistance at high-dose histamine is due to bronchoconstriction alone or to other mechanisms (mucus, edema, hyperemia) that are not reversed by bronchodilators.

Although the title of their paper would seem to be obvious, based on previously published work in humans, their suggested mechanism for the interaction of halothane and aminophylline is not entirely convincing. Nevertheless, I concur with their conclusions that in humans the administration of a β2 agonist is more appropriate than aminophylline in the treatment of bronchospasm during anesthesia and have previously published this recommendation.  

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REFERENCES
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In Reply—We would like to thank Dr. Jones for his thoughtful comments and questions concerning our manuscript. We agree that in order to assume that the lack of effect of aminophylline indeed results from inhibition of catecholamine release by halothane, one must show that additional bronchodilatation is possible beyond that achieved with 1.5 MAC halothane. In a follow-up study we evaluated the efficacy of albuterol during both thiopental-fentanyl anesthesia and during 1.5 MAC halothane anesthesia. Although halothane attenuated histamine-induced bronchoconstriction when compared with the effects of thiopental-fentanyl anesthesia, the administration of albuterol during halothane anesthesia further attenuated the pulmonary response to histamine. These data suggest that although halothane attenuates histamine-induced bronchoconstriction, this attenuation is not complete at 1.5 MAC halothane.

We agree that albuterol is more effective than aminophylline at attenuating histamine-induced bronchoconstriction; regardless of the anesthetic (thiopental-fentanyl or halothane). We further agree based on our current work and previous studies that the administration of a β agonist is the treatment of choice for bronchospasm during inhalational anesthesia.

REFERENCES

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