Correspondence

Responses of Bronchial Smooth Muscle

To the Editor:—In reference to the interesting article by Cook and Chodoff, "Anesthetic Management of an Incompletely Controlled Hyperthyroid Patient for Thyroidectomy" (ANESTHESIOLOGY 33:562, 1970), I would like to inquire about one point. Under "Discussion" the authors state "we completed her adrenergic blockade with phentolamine for the following reasons: . . . 2) Beta blockade alone also leaves unopposed constrictor receptors in the bronchi which could precipitate bronchospasm."

It has been my impression that bronchial smooth muscle responds to adrenergic stimulation (beta receptor) by relaxation and to cholinergic impulses by contraction. (For example, see Koelle, "Neurohumoral Transmission and the Autonomic Nervous System," in Goodman and Gilman: *The Pharmacological Basis of Therapeutics*, 1970, chap. 21, p. 402–441.) I am unaware of any response of bronchial smooth muscle to alpha stimulation or blockade, or to any direct action of phentolamine.

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To the Editor:—I would like to offer the following explanation: 1) It is well established that beta receptors in the bronchi produce bronchial relaxation when stimulated.[1] 2) There is experimental evidence in dogs that there are alpha receptors responsible for bronchoconstriction.[2] 3) Studies of asthmatics have shown increases in airway resistance, wheezing and dyspnea after the administration of propranolol. This response could be completely abolished by the prior administration of atropine. Normal subjects also showed increases in mean airway resistance, but to a much lesser degree.[3]

While alpha receptor stimulation has not been shown to be a significant factor in human bronchoconstriction, there are circumstances when these drugs should be used to prevent possible bronchoconstriction. Our patient had the following potential causes for bronchoconstriction: secretions, unopposed vagal activity (no belladonna drug for premedication), and thiopental for induction. These conditions can produce bronchoconstriction, which does not normally respond to alpha receptor blockade. However, this patient's beta blockade, which would have prevented any bronchodilitation, could have made her sensitive to otherwise insignificant constrictor stimuli, including the possibility of alpha activity.

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