Effect of Pulmonary Sympathetic Blockade on Bronchial Responsiveness

To the Editor.—A recent article by Groeben et al. suggested that high thoracic epidural anesthesia did not alter airway resistance and attenuated the response to inhaled acetylcholine challenge in patients with preexistent bronchial hyperreactiveness. In light of other recently published data, the results of this study are unexpected.

The parasympathetic nerve supply of the lungs arises in the vagus nuclei of the brainstem, from which efferent fibers pass down the vagus nerve to synapse in ganglia within the airway wall. Sympathetic pulmonary innervation originates from the upper six thoracic ganglia. Although human airways appear to lack tonic sympathetic bronchodilator tone, it is believed that sympathetic nerves in the lung act in a neuromodulatory capacity, serving to counteract a cholinergic bronchoconstrictor effect. A situation that produces selective blockade of sympathetic input to the lung therefore would be expected to enhance bronchial responsiveness. Such is the case in cervical spinal cord injury (quadriplegia), in which transection of the cervical spine interrupts sympathetic pulmonary innervation, resulting in unopposed cholinergic airway tone. We have demonstrated the uniform presence of bronchial hyperresponsiveness to inhaled methacholine in subjects with chronic cervical spinal cord injury, who were lifetime nonsmokers without history of respiratory disease predating their injury. Subjects with lesions of the lower thoracic (below T6) and lumbar spine displayed normal bronchial reactivity. Our explanation of these findings is based on unopposed parasympathetic airway innervation is supported by recent animal studies. Using a guinea pig model, Hey et al. induced vagal cholinergic bronchoconstriction by electrical stimulation of structures within the rostral region of the dorsal medulla. Interruption of the bulbospinal sympathetic neuronal pathways by administration of lidocaine directly into the spinal cord at the first cervical level potentiated the bronchoconstrictor response, as did bilateral adrenalectomy. In adrenalectomized animals, injection of lidocaine into the spinal cord or administration of intravenous propranolol further potentiated bronchospasam, thereby suggesting the presence of sympathetic inhibitory neural modulation of electrically induced cholinergic bronchoconstriction independent of circulating catecholamines.

These contradictory findings demonstrate the need for further elucidation of the functional significance of pulmonary sympathetic innervation in bronchial reactivity.

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References


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In Reply.—Dicpinigaitis et al. suggest that the results of our study in humans are unexpected in light of recent published studies in humans with cervical spinal cord transections and animal (guinea pig) studies with lidocaine injections directly into the spinal cord. However, we do not think that these findings are contradictory to ours and would like to offer some alternate explanations.

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