this issue as part of our study, but we felt that the work of Liu et al.\(^2\) had already answered this question definitively.

**References**


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**Pretreatment Intravenous Lidocaine for Intubation of the Asthmatic Patient: More Data Are Needed**

*To the Editor:*—Important issues regarding the use of lidocaine as a pretreatment agent before intubation in patients with reactive airway disease remain unresolved. In the October 1998 issue of Anesthesiology, the use of intravenous lidocaine was recommended to attenuate airway reflexes in patients with bronchial hyperreactivity.\(^1\) This recommendation is based on the finding that lidocaine attenuates reflex bronchoconstriction caused by inhaled histamine. Furthermore, the authors reported no significant change in mean values of forced expiratory volume in 1 s at baseline and after lidocaine administration.

I remain unconvinced that intravenous lidocaine is a safe and effective agent in patients with reactive airway disease. Multiple studies have demonstrated an acute bronchoconstrictor effect by inhaled lidocaine.\(^2-4\) The ability of lidocaine to attenuate histamine-induced bronchoconstriction does not refute the concern that lidocaine may precipitate small airway bronchoconstriction in patients with reactive airway disease. McAlpine and Thomson\(^5\) found no correlation between asthmatic subjects with histamine airway hyperresponsiveness and those who suffered bronchoconstriction produced by inhaled lidocaine. In addition, changes in expiratory flow rates after inhaled lidocaine demonstrate much intersubject variability; use of mean number values can overlook this important intersubject variability. Prakash et al.\(^6\) reported that 3 of 15 stable asthmatic subjects experienced a ≥10% decrease in maximum midexpiratory flow, and 1 of 15 subjects experienced a ≥10% increase in maximum midexpiratory flow. McAlpine and Thomson\(^7\) reported that maximum percent change in forced expiratory volume in one s among stable asthmatic subjects after inhaled lidocaine varied from −42.1 to +28.2. Thus, I am skeptical that attenuation of a marker of bronchial reactivity, such as a histamine challenge, is adequate data to support a recommendation of intravenous lidocaine use in patients with reactive airway disease. Intravenous lidocaine efficacy should be demonstrated in a general population of asthmatic patients, not just a subgroup that demonstrates hyperreactive responsiveness to histamine, and data presentation should address possible intersubject variability.

**References**


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