tion of their patients. If esmolol does not effect its anesthetic action *via* nitrous oxide, did the authors notice a difference in the time to loss of consciousness between the propofol only, and the propofol plus esmolol groups, in the absence of nitrous oxide?

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In Reply: — We appreciate Dr. Baranov and Dr. Bagshaw's constructive comments. The mechanism by which esmolol decreases anesthetic requirements cannot be deduced from our paper. Our goal was to document the existence of this interaction. The balanced-anesthetic technique used in the paper was complex, involving propofol, nitrous oxide, and morphine premedication. At the time, this technique represented the best clinical conditions for demonstrating the effect of esmolol. An interaction between any of the anesthetic components could be hypothesized. The experiment was specifically designed to examine the interaction under conditions approaching steady state. No attempt was made to evaluate differences in anesthetic induction between groups.

Currently, available clinical evidence suggests that esmolol potentiates opioid action. We have found that esmolol has no effect on isoflurane MAC alone but can potentiate MAC reduction by alfentanil. This supports an earlier observation by Stanley *et al.* that patients receiving long-term, propranolol treatment required 25% less fentanyl to produce loss of consciousness.

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