block hypocapnic bronchoconstriction probably is mediated not through an adrenergic mechanism, but by one that is nonspecific.

This study was supported in part by the Ohio Medical Products Division of Airco, Inc. Penthrane was supplied by Abbott Laboratories; Fluothane was supplied by Ayerst Laboratories; and Ethrane was supplied by Ohio Medical Products.

References

1. Patterson RW, Sullivan SF, Malm JR, et al: The effect of halothane on human airway mechanics. ANESTHESIOLOGY 29:900–907, 1968
2. Meloche R, Norlander O, Norden I, et al: Effects of carbon dioxide and halothane on compliance and pulmonary resistance during cardio-pulmonary bypass. Scand J Thorac Cardiovasc Surg 3:69–78, 1969
3. McAslan C, Mima M, Norden I, et al: Effects of halothane and methoxyflurane on pulmonary resistance to gas flow during lung bypass. Scand J Thorac Cardiovasc Surg 5:193–197, 1971
4. Klide AM, Aviado DM: Mechanism for the reduction in pulmonary resistance induced by halothane. J Pharmacol Exp Ther 158: 28–35, 1967
5. Rattenborg CC, Benfield JR, Nigro SL, et al: Differential measurement of pulmonary compliance in dogs. Physiologist 5:199, 1962
6. Severinghaus JW, Swenson EW, Finley TN, et al: Unilateral hypoventilation produced in dogs by occluding one pulmonary artery. J Appl Physiol 16:53–60, 1961
7. Don HF, Robson JG: The mechanics of the respiratory system during anesthesia. ANESTHESIOLOGY 26:168–178, 1965
8. Rattenborg CC, Holaday DA: Constant flow inflation of the lungs, theoretical analysis. Acta Anaesthesiol Scand 23:211–223, 1967
9. Saidman LJ, Eger EI, Munson ES, et al: Minimum alveolar concentrations of methoxyflurane, halothane, ether and cyclopropane in man: Correlation with theories of anesthesia. ANESTHESIOLOGY 28:994–1002, 1967
10. Gion H, Saidman LJ: The minimum alveolar concentration of enflurane in man. ANESTHESIOLOGY 35:361–364, 1971
11. Hickey RF, Graf PD, Nadel JA, et al: The effects of halothane and cyclopropane on total pulmonary resistance in the dog. ANESTHESIOLOGY 31:334–343, 1969
12. Aviado DM: Regulation of bronchomotor tone during anesthesia. ANESTHESIOLOGY 42:68–80, 1975
13. Fletcher SW, Flacke W, Alper MH: The actions of general anesthetic agents on tracheal smooth muscle. ANESTHESIOLOGY 29:517–522, 1968
14. Dollery CT, Paterson JW, Conolly ME: Clinical pharmacology of beta-receptor blocking drugs. Clin Pharmacol Ther 10:765–799, 1969

Propranolol

DANGERS OF PROPRANOLOL WITHDRAWAL Because of the potential dangers of anesthesia in the presence of *beta*-adrenergic blockade, some clinicians have advocated that propranolol be discontinued prior to anesthesia and operation. The authors call attention to the possible adverse effects of such an endeavor. Six patients with severe angina had been treated with 240 mg/day of propranolol. In all patients, the frequency of anginal pain had decreased substantially since therapy had been instituted. Propranolol therapy was abruptly discontinued in four patients because of research protocols, in one patient because of lack of availability, and in one patient because of scheduled angiography. In all six patients frequent, prolonged chest pain occurred at rest immediately after propranolol was discontinued. The pattern of pain was distinctly different from that experienced either before or during propranolol therapy. After 2–21 days of continuing unstable angina, three patients had acute myocardial infarction, while a fourth suddenly died. That angina at rest and nocturnal angina appeared in patients never previously experiencing such symptoms, as well as the increase in frequency and severity of pain, suggests that this represented a true rebound phenomenon rather than symptom reappearance. The authors point out that these findings should be considered when abrupt withdrawal of propranolol therapy is contemplated. *(Alderman EW, Coltart DJ, Wettach GE, et al.: Coronary Artery Syndromes after Sudden Propranolol Withdrawal. Ann Intern Med 81: 625–627, 1974.)*