cludes high-pressure alarms, which are designed solely to reveal the existence of a dangerous high pressure in the system but not to reveal any other hazardous condition that may or may not be accompanied by high pressure.

PETER J. SCHREIBER
*President*
*North American Drager*
*148B Quarry Road*
*Telford, Pennsylvania 18969*

Anesthesiology
63:233, 1985

## Antiarrhythmic Effect of Verapamil May Be Independent of Calcium Channel Blockade

*To the Editor:*—Kroll and Knight demonstrated that verapamil, halothane, enflurane, and isoflurane each significantly reduced ventricular fibrillation in an occlusion–reperfusion arrhythmia model.[1] Because of verapamil's well-defined action as a calcium entry blocker and the ability of the volatile anesthetics to modulate calcium ion translocation, they suggested that blockade of calcium channels was the probable mechanism for each drug's antifibrillatory effect. Lynch's accompanying editorial[2] emphasizes that volatile anesthetics may exert their antiarrythmic effect by actions other than by calcium channel blockade. However, verapamil's heterogeneity, too, should not be overlooked, for it possesses many effects, other than calcium channel blockade, which could be antiarrhythmic in this setting.[3] Kroll and Knight have convincingly discounted some of these properties (*e.g.*, fast-channel blockade) for verapamil's antiarrhythmic action, but others, including its alpha-adrenergic blocking properties,[4] were not addressed. Using a feline occlusion–reperfusion model, Corr's group have established the antiarrhythmic effect of $alpha_1$ adrenergic blockade.[5] Consistent with this pharmacologic effect, the myocardial $alpha_1$ adrenoceptors density was significantly increased during the occlusion and early reperfusion period.[6] Verapamil also has been shown to raise the arrhythmia threshold in a canine halothane–epinephrine arrhythmia model[7] in which the mediating mechanism is predominantly the alpha adrenoceptor.[8]

Thus, in Kroll and Knight's study, the antiarrhythmic effects of verapamil and volatile anesthetics may be operating through entirely different mechanisms, *both* independent of calcium channel blockade.

SAMUEL METZ, M.D.
*Fellow in Cardiovascular Anesthesia*
*Texas Heart Institute*
*Houston, Texas*

MERVYN MAZE, M.D.
*Assistant Professor of Anesthesia*
*Stanford University School of Medicine*
*Stanford, California*

### REFERENCES

1. Kroll DA, Knight PR: Antifibrillatory effects of volatile anesthetics in acute occlusion–reperfusion arrhythmias. ANESTHESIOLOGY 61:657–661, 1984
2. Lynch C: Are volatile anesthetics really calcium entry blockers? ANESTHESIOLOGY 61:644–646, 1984
3. Thandroyen FT: Protective action of calcium channel antagonist agents against ventricular fibrillation in the isolated perfused rat heart. J Mol Cell Cardiol 14:21–32, 1982
4. Karliner JS, Motulsky HJ, Dunlop J, Brown JH, Insel PA: Verapamil competitively inhibits $alpha_1$-adrenergic and muscarinic but not beta-adrenergic receptors in rat myocardium. J Cardiovasc Pharmacol 4:515–520, 1982.
5. Sheridan DJ, Penkoske PA, Sobel BE, Corr PB: Alpha-adrenergic contributions to dysrhythmia during myocardial ischemia and reperfusion in cats. J Clin Invest 65:161–171, 1980
6. Corr PB, Shayman JA, Kramer JB, Kipnis RJ: Increased alpha adrenergic receptors in ischemic cat myocardium. J Clin Invest 67:1232–1236, 1981
7. Kapur PA, Flacke WE: Epinephrine-induced arrhythmias and cardiovascular function after verapamil during halothane anesthesia in the dog. ANESTHESIOLOGY 55:218–225, 1981
8. Maze M, Smith CM: Identification of receptor mechanism mediating epinephrine-induced arrhythmias during halothane anesthesia in the dog. ANESTHESIOLOGY 59:322–326, 1983

*(Accepted for publication March 15, 1985.)*

Anesthesiology
63:233–234, 1985

## The Diagnosis of "Junctional Rhythms" with Halogenated Anesthetics

*To the Editor:*— We have read "Successful Treatment of Accelerated Junctional Rhythm with Propranolol: Possible Role of Sympathetic Stimulation in the Genesis of this Rhythm Disturbance"[1] with interest.

We question whether this may be a case of isorhythmic dissociation (ID)[2,3] rather than the accelerated nodal rhythm reported, since, as pointed out by Sethna *et al.*,[4] ID is common during inhalational anesthesia but often