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.

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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.

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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

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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

improperly diagnosed as junctional rhythm. During periods of P wave disappearance, ID is difficult to distinguish from nodal rhythms. Furthermore, heart rate has been noted to increase on conversion from sinus rhythm to ID.*

ID may be defined as a type of A-V nodal dissociation, whereby the S-A and A-V nodes fire at almost identical rates, without conduction across the A-V node. In ID the upright P wave will be seen to gradually merge with the QRS complex, whereas in a nodal rhythm the P wave will change its configuration or be lost within the QRS complex. As noted by Sethna *et al.*[4] "(in ID,) if the moment of dissociation is missed . . . the pattern is one of QRS complexes without visible P waves and may be misread as A-V nodal rhythm." With continuous observation, the decreasing P-R interval will be noted.

There is evidence that ID may be an extremely frequent occurrence when halogenated anesthetic agents are used. In one study of normal healthy volunteers under isocapnic enflurane anesthesia without surgery, Calverley *et al.*[5] demonstrated ID in five out of 12 subjects. No case of junctional rhythm was noted. We have noted many such occurrences at our institution while using halothane, isoflurane, and enflurane. Boba[6] has noted what appears to be ID with methoxyflurane.

The etiology of ID remains unclear, as does its treatment. Breslow *et al.*[1] suggest that light anesthesia may be an implicating factor in the arrhythmia that they report. However, Laver and Turndorf[7] noted that increasing the halothane concentration from 0.3 to 1.0% leads to the development of this arrhythmia. Also, as noted above, this disturbance has been seen both in the presence and absence of surgical stimulation.

Finally, we were surprised at the suggestion of the authors that a switch from halothane to enflurane during surgery would have restored normal sinus rhythm in light of the report of Calverley *et al.*[5]

An increased awareness of the occurrence of ID during inhalational anesthesia and continuous monitoring of ECG may lead to the correct diagnosis of this common, but often misdiagnosed, rhythm disturbance.

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REFERENCES

1. Breslow MJ, Evers AS, Lebowitz P: Successful treatment of accelerated junctional rhythm with propranolol: Possible role of sympathetic stimulation in the genesis of this rhythm disturbance. ANESTHESIOLOGY 61:180–182, 1985
2. Schubart AF, Marriott HJL, Gorten RJ: Isorhythmic dissociation: Atrio-ventricular dissociation with synchronization. Am J Med 24:209–214, 1958
3. Marriott HJL: Interactions between atria and ventricles during interference-dissociation and complete A-V block. Am Heart J 53:884–889, 1957
4. Sethna DH, Deboer GE, Millar RA: Observations on "Junctional Rhythms" during anaesthesia. Br J Anaesth 56:924–925, 1984
5. Calverley RK, Ty Smith N, Prys-Roberts C, Eger EI II, Jones CW: Cardiovascular effects of enflurane anesthesia during controlled ventilation in man. Anesth Analg 57:619–628, 1978
6. Boba A: Significant effects on the blood pressure of an apparently trivial atrial dysrhythmia. ANESTHESIOLOGY 48:282–283, 1978
7. Laver MB, Turndorf H: Atrial activity and systemic blood pressure during anesthesia in man. Circulation 28:63–71, 1963

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* Ty Smith N, Calverley RK, Jones CW, Prys-Roberts C, Eger EI II: The hemodynamic impact of atrial arrhythmias during enflurane anesthesia in man. Scientific Abstracts of the 1977 Meeting of the American Society of Anesthesiologists, pp 99–100.

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Action of Verapamil at the Neuromuscular Junction: Prejunctional or Postjunctional?

To the Editor:—I read with great interest, in the Correspondence section of ANESTHESIOLOGY, the controversy concerning the site of action of verapamil at the neuromuscular junction.[1,2] Durant *et al.* showed in their original report that verapamil potentiates the neuromuscular block of pancuronium and succinylcholine and state that this effect is not centered on the muscle fiber itself.[3] Foldes, however, took issue with this con-