

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 editorial² emphasizes that volatile anesthetics may exert their antiarrhythmic 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 alpha₁ adrenergic blockade.⁵ Consistent with this pharmacologic effect, the myocardial alpha₁ 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 model⁷ 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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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

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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. Boba⁶ 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 Turndorf⁷ 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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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-