between the placebo and metoprolol groups. When the data for the placebo group were analyzed with respect to cardiac dysrhythmias, no difference was found between the concentration of norepinephrine or epinephrine in the dysrhythmic group compared to those in whom no dysrhythmias occurred (Table III).

There are two main major conclusions from the study. First, although catecholamine levels increase significantly during oral surgery there appears to be no simple relationship between plasma catecholamine concentration and dysrhythmic episodes. Second, a single oral dose of metoprolol 2 hours before surgery substantially decreases the incidence of serious cardiac dysrhythmias. Metoprolol is cardioselective and clinical studies have demonstrated its efficacy in many cardiac dysrhythmias.[6] The results of the present study suggest that this agent might offer a simple therapy to reduce potentially dangerous cardiac events during oral surgical procedures.

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A case for intra-atrial Wenckebach heart block during atrial flutter

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We present an example of an unusual dysrhythmia that may be explained by a variety of possible mechanisms, including intra-atrial Wenckebach heart block. Although Mobitz I block has been demonstrated at all levels within the heart, we are aware of only one other case report postulating intra-atrial Wenckebach block to explain a spontaneously occurring dysrhythmia not produced by atrial pacing.[1]

The patient is a 45-year-old white male renal transplant recipient with reactive airway disease, coronary artery disease, recurrent congestive heart failure, and recurrent supraventricular dysrhythmias, including supraventricular tachycardia, atrial fibrillation, atrial flutter, and frequent atrial premature beats. He was admitted on Dec. 19, 1980, with palpitations and dyspnea. While the patient was taking oral aminophylline, 200 mg. every 8 hours; oral metoprolol, 12.5 mg twice a day; oral digoxin, 0.5 mg four times a day; oral furosemide, 120 mg twice a day; oral metolazone, 5 mg twice a day; oral isosorbide dinitrate, 10 mg every 6 hours; and sublingual nitroglycerin as needed, his pulse was 104 bpm and "regular," blood pressure was 150/80 mm Hg, and respirations were 24/min. There was no neck vein distension at 30 degrees. The lung examination was negative. Cardiac examination revealed lateral displacement of the point of maximal impulse and a grade 3/6 holosystolic apical murmur radiating to the axilla. Admission electrolytes including calcium were normal except for a CO_2 of 18 mEq/L. Blood urea nitrogen and creatinine were 80 mg/dl and 5.5 mg/dl, respectively, consistent with the patient's usual values for these parameters. White blood count was 4800/mm.[3] Hemoglobin and hematocrit values were 9.8 gm/L and 29.6%, respectively. A digoxin level 2 days prior to admission was 3.6 mg/ml. The patients's admission ECG revealed the rhythm discussed below, as well as left ventricular hypertrophy with secondary ST-T segment abnormalities and a possible old inferior wall myocardial infarction.

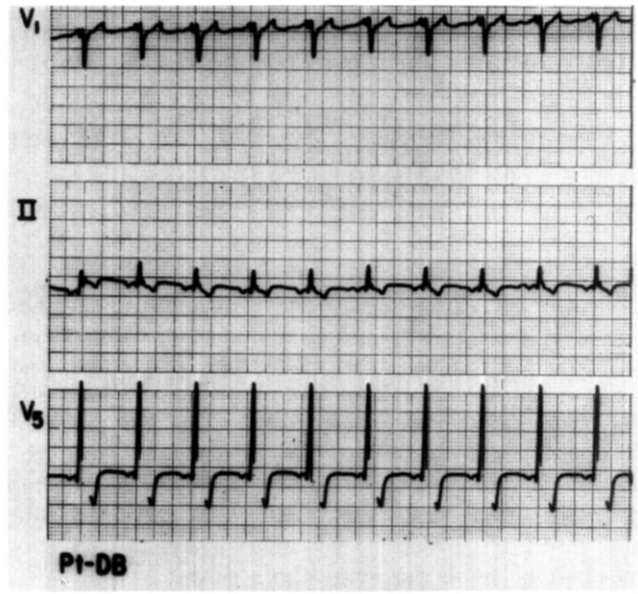

Fig. 1. Rhythm strip showing dysrhythmia simultaneously in leads V_1, II, and V_5.

P waves were negative in leads I, II, III, and aV_F before the QRS complex, with a PR interval of 0.11; a P wave also follows each QRS complex but is partially obscured by the T wave (Fig. 1). The P wave following the QRS complex (P_2) appears to have the same morphology as that preceding the QRS complex (P_1); but since P_2 is partially

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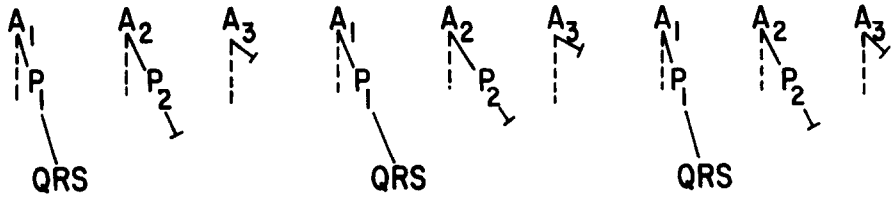

Fig. 2. Schematic diagram of mechanism of dysrhythmia if P waves are identical. *A* = Atrial focus firing at a rate of 291 bpm, *not* visible on the surface ECG; *P* = P wave, visible on the surface ECG; *QRS* = ventricular depolarization visible on the surface ECG. Note that the intervals between atrial impulses (A_1 to A_2, A_2 to A_3, and A_3 to A_1) are equal. Intra-atrial conduction, manifested by the time interval between A and P, "progressively" lengthens, so that A_2 to P_2 is longer than A_1 to P_1, there is intra-atrial block after A_3, and the next cycle begins with a short interval from A_1 to P_1, etc. The diagram also shows the QRS morphology after P_1 only, with block somewhere between P_2 and the ventricles. As on the surface ECG, the QRS-P_2 interval is constant; the interval from P_1 to P_2 is also constant, and it is shorter than P_2 to P_1, which is constant.

obscured by the T wave, it is uncertain whether its morphology is identical to that of P_1. If the morphologies are not identical, it could be assumed that the initiating site and/or pattern of atrial depolarization is different for P_1 and P_2. If the morphologies of P_1 and P_2 are identical, it could be assumed that the initiating sites and patterns of atrial depolarization are identical.

Given this ambiguity of the character of the atrial depolarizations, we hypothesize the following possible mechanisms of the abnormal rhythm: (1) If the P wave morphologies are different, (a) a "low" (nonsinus) atrial focus occurs at a rate of 97 bpm, causing P_1 and the subsequent QRS morphology, with retrograde depolarization of the atrium after each QRS complex through a reentry pathway distal to the atrium (the atrioventricular node or an accessory ventriculoatrial pathway); (b) a junctional tachycardia occurs at a rate of 97 bpm, with retrograde conduction along both fast and slower atrioventricular nodal pathways, resulting in waves P_1 and P_2, respectively; or (c) atrial bigeminy occurs because of reentry within the atrium, with P_2 not leading to depolarization of the ventricles, because of refractoriness somewhere between the atrium and the ventricles. (2) If the P wave morphologies are identical, then atrial flutter occurs at a rate of 291 bpm (three times the ventricular rate), with 3:2 intra-atrial Wenckebach heart block and 2:1 heart block distal to the atria because of refractoriness distal to the atria at the time of P_2 (Fig. 2).

Without intra-atrial recordings, we cannot definitively characterized the mechanism of this dysrhythmia. Those outlined seem feasible, and if P wave morphologies are identical, intra-atrial Wenckebach rhythm seems likely. The presumed atrial rate of 291 bpm is compatible with the primary rhythm's being atrial flutter. Wenckebach rhythm in the atrium between an electrical stimulus site and another region of the atrium is not uncommon in electrophysiologic studies and in that context has been described in the literature as early as 1918 to 1920 by Lewis[2] and more recently by Castellanos et al.[3] and Narula et al.[4] In addition, Schamroth[5] states that it may occur with automatic atrial tachycardias, although he does not cite examples. The present case supports the concept of a Wenckebach form of exit block from an atrial focus (either microreentry or automatic) distinct from the sinus node, in a nonelectrically stimulated atrium.

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Ventricular fibrillation with spontaneous reversion on ambulatory ECG in the absence of heart disease

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There is increased interest concerning the influence of the central nervous system in the genesis of ventricular ectopic beats (VEBs), and there is evidence that a high level of

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