Paradoxic Acceleration of Ventricular Rate after Therapy with Lidocaine and Ajmaline*

Findings in Two Patients with Supraventricular Tachyarrhythmia

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In two patients with supraventricular tachyarrhythmias with atrioventricular block, therapy with lidocaine and ajmaline decreased the atrioventricular block and caused paradoxic acceleration of the ventricular rate. Appropriate treatment of this hazardous result of therapy with antiarrhythmic drugs is reviewed.

Paradoxic acceleration of the ventricular rate is a well-known risk of drug therapy for rapid supraventricular arrhythmias with atrioventricular block. The classic example is seen in therapy with quinidine for atrial flutter. As quinidine slows the atrial rate and blocks the vagal effect on the atrioventricular node, the number of impulses reaching the atrioventricular node is reduced from a rate between 300 and 350 per minute to between 180 and 200 per minute, a rate which the atrioventricular node can easily handle, with a consequent acceleration from 150 to 200 per minute. Administration of digitalis before quinidine therapy is therefore recommended.1 2

The occurrence of paradoxic acceleration with drugs other than quinidine is not well known. We report two cases in which paradoxic acceleration resulted from therapy with lidocaine and ajmaline. One patient was successfully treated with verapamil and the second with a β-adrenergic blocking agent.

Case Reports

Case 1

A 72-year-old man with chronic diffuse interstitial nephritis and uremia was admitted to the coronary care unit because of suspected digitalis intoxication. He had received digoxin (0.125 mg/day) for congestive heart failure following a previous myocardial infarction. For an undetermined reason, the patient had increased the dose to 0.25 mg/day two to three weeks prior to admission.

The patient was seen in the emergency room on Jan 29, 1975 because of shortness of breath. Examination revealed wheezes and rhonchi, but there was no clear-cut evidence of heart failure. The electrocardiogram showed atrial tachy-
is able to handle impulses up to a rate of about 200 impulses per minute. Atrial arrhythmias with a rate higher than this are usually associated with 2:1 or varying atrioventricular block. When digitalis "protects" the atrioventricular node, atrioventricular block will occur at even slower rates. Acceleration of the ventricular rate

**Figure 1.** Electrocardiograms with paper speed of 25 mm/sec (case 1). A, Supraventricular tachycardia with atrial rate (AR) of 263 impulses per minute and ventricular rate (VR) of 118 beats per minute. Beats 6 and 12 and penultimate beat probably represented conducted beats and were misinterpreted as ventricular premature beats. B and C, After administration of 100 mg of lidocaine, prolonged bursts of 1:1 atrioventricular conduction occurred, with atrial and ventricular rates of 190 to 200 impulses per minute, L₁, lead 1; and L₂, lead 2.

**Figure 2.** Electrocardiograms (case 1). A, return of atrial rate (AR) to 210 impulses per minute and 2:1 atrioventricular conduction. B and C, Shortly after administration of 50 mg of ajmaline, atrial rate slows again to 175 impulses per minute. Arrows indicate atrial P waves. D, Atrial rate slows to 150 impulses per minute, at which point persistent 1:1 atrioventricular conduction ensues. E, Administration of verapamil reestablishes 2:1 atrioventricular block. L₁, lead 1.
can result from either slowing of the atrial rate, a decrease in the atrioventricular block, or both. The primary mechanism of quinidine-induced ventricular acceleration is the slowing of the atrial rate, but there is some evidence that a vagolytic effect on the atrioventricular node may also be partially responsible.1,4 The present report demonstrates that the same acceleration of the ventricles can be produced by administration of lidocaine and ajmaline, and that therapy with lidocaine may be capable of enhancing atrioventricular conduction even when the atrioventricular node is presumably protected by digitalis. Marriott and Bieza5 have reported a case of atrial flutter in which the ventricular rate accelerated from 165 to 265 beats per minute following administration of lidocaine, and similar cases have also been reported.4 In our first patient, bursts of ventricular activity at 200 beats per minute also appeared with a decrease in atrial rate from 230 to 200 impulses per minute. Rosen et al6 observed shortening of the P-His interval in four of ten patients after administration of lidocaine. The effects of lidocaine therapy on atrioventricular nodal fibers with a toxic reaction to digitalis have not been studied in detail and certainly merit further attention in the light of our experience with patient 1. Therapy with lidocaine is capable of improving atrioventricular conduction even in patients receiving digitalis,5 and our case suggests a similar effect in digitalis-induced paroxysmal atrial tachycardia with 2:1 block.

The bursts of rapid ventricular rate in the first patient became more frequent and finally changed to a sustained ventricular rate of 150 beats per minute under the influence of ajmaline. In the second patient, acceleration occurred just a few moments after administration of ajmaline. Ajmaline shares several properties with quinidine in its effects on cardiac fibers.6,7 It decreases the maximum rate of depolarization and the velocity of conduction in Purkinje's fibers,6 and Wells and Durre7 observed prolongation of the His-ventricle interval by ajmaline therapy in man. On the other hand, as with quinidine and procaine amide hydrochloride,8,9,10 administration of ajmaline does not always lengthen the effective refractory period of the atrioventricular node.9 It, thus, is predictable that ajmaline therapy, by slowing the velocity of conduction and the rate in the atrium without simultaneously increasing the effective refractory period of the atrioventricular node, can produce the same paradoxical acceleration of the ventricles as quinidine and lidocaine.

We concur with others4,5 that lidocaine therapy should be used for the treatment of ventricular premature beats in the presence of supraventricular tachyarrhythmias with atrioventricular block only if absolutely necessary. Our experience dictates the same caution with regard to ajmaline. The acceleration of the ventricular rate as seen here is potentially dangerous and requires prompt treatment. Administration of digitalis takes a minimum of ten minutes to be effective, or it may be contraindicated, as in case 1. Beta-adrenergic blockers may or may not be effective. It is, therefore, of interest to note that verapamil therapy seems to be rapidly effective. Administration of verapamil rapidly increases the effective refractory period of the atrioventricular node and slows conduction of all atrial impulses through the atrioventricular node.11 Verapamil essentially fulfills the function in the atrioventricular node that is required of digitalis glycosides, acts more promptly, and is not contraindicated in the presence of digitalis intoxication.

**References**


**Disseminated Coccidioidomycosis with Pericarditis**

**Successful Treatment with Amphotericin B**

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Pericarditis with effusion can occur as a complication of disseminated coccidioidomycosis. Information on management of this condition is very scanty in the medical literature. One case is described in detail. Early diagnosis and appropriate therapy with amphotericin B have been emphasized as keys to success in treating this condition.

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