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Intravenous Amiodarone Versus Verapamil for Acute Conversion of Paroxysmal Atrial Fibrillation to Sinus Rhythm

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Amiodarone and verapamil are well-known antiarrhythmic drugs used for treatment of ventricular and supraventricular arrhythmias. Although verapamil is the drug of choice for control of the atrioventricular node, it has also been reported to terminate atrial fibrillation.[1–3] Amiodarone has been used extensively for drug-refractory ventricular tachycardia but seldom for termination of paroxysmal atrial fibrillation.[4–6] To our knowledge, no comparative study with amiodarone and verapamil has been reported. Because of this, we compared the efficacy of intravenous amiodarone versus verapamil for conversion of paroxysmal atrial fibrillation to sinus rhythm in a single-blind randomized study.

The patient population consisted of 24 consecutive patients with paroxysmal atrial fibrillation (15 men and 9 women aged 71 ± 9.6 years, range 51 to 85). The duration of arrhythmia ranged from 20 minutes to 48 hours at entry to the study. The mean ventricular rate was 125 ± 27 beats/min. There was no statistically significant difference in age, sex, heart rate, duration of arrhythmia before arrival and incidence of different heart disease between the amiodarone and verapamil groups. Exclusion criteria were known or suspected conduction disturbances, including preexcitation; sick sinus syndrome; hyperthyroidism; concomitant therapy with antiarrhythmic drugs; arrhythmia-related systemic arterial hypotension; and any sign of heart failure.

Medical history, clinical examination, routine laboratory testing and a 12-lead electrocardiogram were performed. Informed consent was obtained and patients were treated with either amiodarone (5 mg/kg body weight intravenously over a 3-minute period) or verapamil (0.075 mg/kg intravenously over a 1-minute period, repeated after 10 minutes). Whether the patient received amiodarone or verapamil was determined randomly. Treatment was considered successful if conversion occurred within 3 hours after administration of the drug. An alternative drug was given and observation time was prolonged for an additional 3 hours, but only if the initially randomized drug failed to convert the atrial rhythm for a 3-hour period. Each patient's rhythm was observed on a monitor and recorded on a tape (DMI Holter recorder) during observation time or until conversion took place. Systemic blood pressure was measured noninvasively every 5 minutes and in cases of hypotension, every minute. The protocol was approved by the state ethics committee. The data were analyzed by means of Student's t test for unpaired variables and Fisher's test for attributive variables.

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TABLE I Rate of Conversion to Sinus Rhythm after Antiarrhythmic Treatment

	Atrial Fibrillation	
	No.	%
Amiodarone	10/13	77*
Verapamil	0/11	
Amiodarone/verapamil	1/3	
Verapamil/amiodarone	7/11	64
Total amiodarone	17/24	71*
Total verapamil	1/14	7

* $p < 0.001$ vs verapamil.

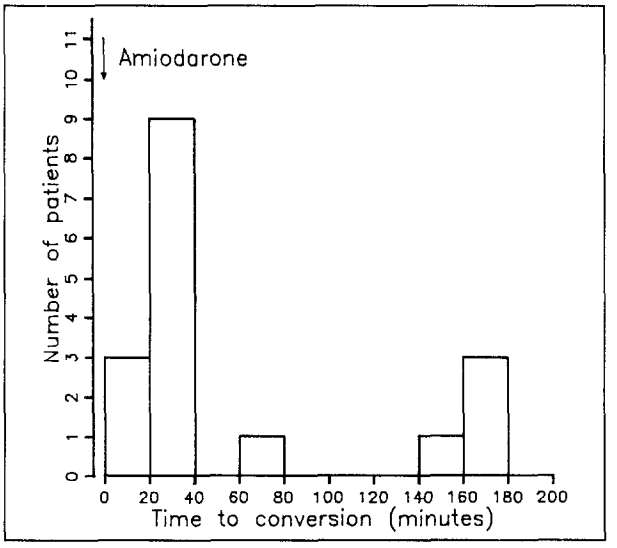

FIGURE 1. Time to conversion of paroxysmal atrial fibrillation to sinus rhythm after treatment with amiodarone.

None of the 11 patients who were initially given verapamil converted to sinus rhythm. However, 77% (10 of 13) of patients who initially received amiodarone converted to sinus rhythm ($p < 0.001$). Amiodarone also seems to be more effective as a second drug but the difference is not statistically significant (Table I). The conversion occurred 10 to 175 minutes after administration of amiodarone (Figure 1). At the time of conversion a variety of electrocardiographic manifestations was observed (ventricular premature beats, nodal premature beats, transient first-degree atrioventricular block, transient atrial flutter, pause of up to 1.1 seconds); none of these manifestations showed a malignant character or were noticed by the patients. Marked adverse effects occurred in 1 patient treated with verapamil (slowing of the ventricular response to 45 beats/min and transitory decrease of systemic arterial pressure from 150/90 to 85/65 mm Hg, which lasted about 5 minutes) and in 1 patient treated with amiodarone (a decrease of systemic arterial pressure from 140/90 to 80/60 mm Hg without bradycardia, which lasted about 4 minutes).

Despite differences in electrophysiologic profile, intravenous amiodarone and verapamil are both used for treatment of patients with paroxysmal atrial fibrillation to slow ventricular response or to promote conversion to sinus rhythm.[1-6] The antifibrillatory effect of intravenous amiodarone has already been demonstrated in animal experiments[7] and may be due to an increase in atrial action potential and the duration of an effective refractory period.[5,6] Verapamil might promote conversion by a possible primary antifibrillatory effect or by an improvement of hemodynamic state due to slowing of the heart rate.[8]

The results of our study show that amiodarone is more effective for conversion than verapamil using the usually recommended and previously reported doses.[1,2,5,6] The overall success rate of amiodarone was 71%, which is better than previously reported success rates.[4-6] The reason for this might be a shorter duration of arrhythmia in our patients (<48 hours) and a longer observation time. The antifibrillatory action of verapamil has not been proved in our study.

The side effects of both drugs, with respect to contraindications, have been rare and have not required discontinuation or additional treatment.

In conclusion, we consider intravenous amiodarone as an effective and safe antiarrhythmic agent in promoting conversion of paroxysmal atrial fibrillation to sinus rhythm.

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Effects of Nicardipine on Left Ventricular Dimensions and Hemodynamics in Systemic Hypertension

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Nicardipine is a new calcium antagonist with potent vasodilatory action.[1] The improvement of cardiac hemodynamics in animals[2] is 1 example of the many promising properties of this drug, which will expectedly lead to its increasing use for the treatment of many cardiovascular disorders, ranging from hypertension to heart failure and from coronary to peripheral vascular disease. Preliminary human trials have also demonstrated an increase of the cardiac pumping ability in subjects with normotension,[3] hypertension,[4] heart failure[5] and coronary artery disease,[6] but most of them have evaluated the short-term effects occurring immediately after the parenteral administration of nicardipine. The aim of this echocardiographic study was to determine the influence of long-term oral nicardipine therapy on cardiac dimensions and hemodynamics in patients with mild to moderate essential hypertension.

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Eighteen patients (4 men, 14 women) with the following characteristics were selected: age (mean ± standard error) 48 ± 2 years (range 33 to 59), hypertensive for 36 ± 11 months, without any systemic, metabolic or endocrinologic disease or evidence of major target organ impairment and previously shown to have no cardiovascular disorder other than mild (10 patients, diastolic pressure <105 mm Hg) to moderate (8 patients, diastolic pressure <115 mm Hg) essential hypertension. The prerequisites for acceptance were a supine resting blood pressure ≥140/95 mm Hg (diastolic blood pressure <115 mm Hg) on 3 separate occasions and the elimination of secondary hypertension by routine screening. The supine resting blood pressure at the beginning of the trial was 161 ± 4/104 ± 2 mm Hg. Seven patients had never been treated for hypertension and the rest had tried various antihypertensives. Informed consent was obtained from all patients.

The protocol was approved by the hospital ethical committee. All medication was stopped 2 weeks before the placebo-controlled trial. The subjects received placebo (identical in appearance to nicardipine) in the first 2