ACUTE TERMINATION OF CARDIAC ARRHYTHMIAS WITH INTRAVENOUS DISOPYRAMIDE

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SYNOPSIS

The acute effects of intravenous disopyramide ("Rhythmodan") given as a single bolus dose of 1-2 mg/kg body weight over a 5 minute period was assessed in 39 patients with a wide variety of cardiac arrhythmias. Six out of 8 patients (75%) with ventricular tachycardia were converted to sinus rhythm. Of the 15 patients with frequent ventricular ectopic beats of more than 10/minute, 11 (73%) had total suppression, whilst 2 (13%) had their PVC's reduced to less than 10% of their previous frequency. All the 4 cases of frequent atrial ectopic beats and the 3 cases with paroxysmal atrial tachycardi showed complete suppression of the arrhythmia. None of the six cases of atrial flutter had any response. All 3 cases with the Wolff Parkinson White Syndrome had their conduction abnormality abolished following intravenous disopyramide. Two of these 3 patients presented concomitantly with atrial fibrillation and this arrhythmia was abolished in 1 patient.

Only 1 patient who had congestive heart failure presented with complications and he had hypotension and slow junctional rhythm following intravenous disopyramide both of which responded to an intravenous isoprenaline infusion.

We conclude that intravenous disopyramide is a very useful anti-arrhythmic agent and is highly effective for the suppression of ventricular tachycardia, ventricular and atrial ectopic beats and for normalization of the WPW conduction abnormality.

INTRODUCTION

Disopyramide is a relatively new anti-arrhythmic agent which has been shown to be effective in suppressing premature ectopic beats and tachycardias of both supraventricular and ventricular origin (1, 2, 3). It possesses properties similar to quinidine and procainamide in decreasing cardiac muscle automatically and conduction velocity (4, 5). Because of its direct depressant action on myocardial function, its use is contraindicated in patients with congestive cardiac failure or cardiogenic shock. We report in this paper the results of an open trial of intravenous disopyramide ("Rhythmodan") to study its effects in a wide variety of cardiac arrhythmias.

PATIENTS AND METHODS

Intravenous disopyramide ("Rhythmodan") was given to 39 patients. The ages of these patients ranged from 27 to 65 years and there were 33 males and six females. Table I lists their underlying cardiac diseases. Thirteen patients had a previous myocardial infarction, 11 had acute myocardial infarction, 2 had unstable angina, 6 had mitral stenosis, 3 had cor pulmonale, 3 had Wolff-Parkinson-White Syndrome and 1 patient had an idiopathic complete heart block complicated by recurrent ventricular tachycardia.

Intravenous disopyramide was given as a single bolus dose of 1 — 2 mg/kg. body weight diluted in 20 mls of sterile water over a 5-minute period in the coronary care unit. Continuous ECG monitoring was done and frequent blood pressure measurements were made using a standard mercurial sphygmomanometer.

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RESULTS

Table II summarizes the results of the intravenous injection of disopyramide. Six out of eight patients (75%) with ventricular tachycardia were converted to sinus rhythm following I.V. disopyramide. Of the 15 patients with frequent ventricular ectopic beats of more than 10 per minute, 11 had total suppression, whilst 2 had their ectopic beats reduced to less than 10% of their previous frequency.

All the 4 cases of frequent premature atrial ectopic beats and the 3 cases with paroxysmal atrial tachycardia showed complete suppression of the arrhythmia with I.V. disopyramide. None of the six cases with atrial flutter were converted to sinus rhythm.

Table 1
Aetiological Diagnosis of Patients

Old myocardial infarction	13
Acute myocardial infarction	11
Unstable angina	2
Mitral stenosis	6
Cor pulmonale	3
Wolff-Parkinson-White syndrome	3
Idiopathic complete heart block with recurrent ventricular tachycardia	1
Total	39

Two of the 3 cases of Wolff-Parkinson-White (WPW) syndrome presented with rapid atrial fibrillation. In both cases the WPW conduction pattern was abolished by I.V. disopyramide and the ventricular rate was also considerably decreased. One of the two patients with atrial fibrillation was converted to sinus rhythm as well. The third patient with WPW syndrome had such a short PR interval that he was initially misdiagnosed as having ventricular tachycardia. Intravenous disopyramide normalised the conduction abnormality.

A transient but clinically insignificant fall of systolic blood pressure of less than 20 mmHg occurred in 5 patients. However, one patient with congestive cardiac failure developed severe hypotension and bradycardia due to junctional rhythm following I.V. disopyramide. Fortunately, both the bradycardia and hypotension improved with intravenous isoprenaline.

DISCUSSION

Up to recently most of the published reports on disopyramide have related to its efficacy when given as an oral agent. In recent years however several studies have confirmed that this agent is also highly effective when given intravenously (1, 2, 6, 7, 8). For example Deano et al in 1977 reported the following success rate with intravenous disopyramide — conversion to sinus rhythm in 38% of patients with atrial flutter, 20% with atrial fibrillation, 33% with atrial tachycardia, 50% with sustained ventricular tachycardia and 75% with non-sustained ventricular tachycardia. In 18 (78%) of their patients with frequent ventricular ectopic beats, greater than 50% suppression was achieved (1). In a multicentre trial that was carried out in Australia by Tonkin et al, the

IV Disopyramide in Cardiac Arrhythmias (N=39)

	Response	%
Ventricular tachycardia	6/8	75
Frequent PVC's :		
Total suppression	11/15	73
90% suppression	2/15	13
No response	2/15	13
Frequent PAC's:		
Total suppression	4/4	100
Atrial tachycardia	3/3	100
Atrial flutter	0/6	0
WPW conduction	3/3	100

Side-Effects : Severe hypotension and bradycardia in 1 patient

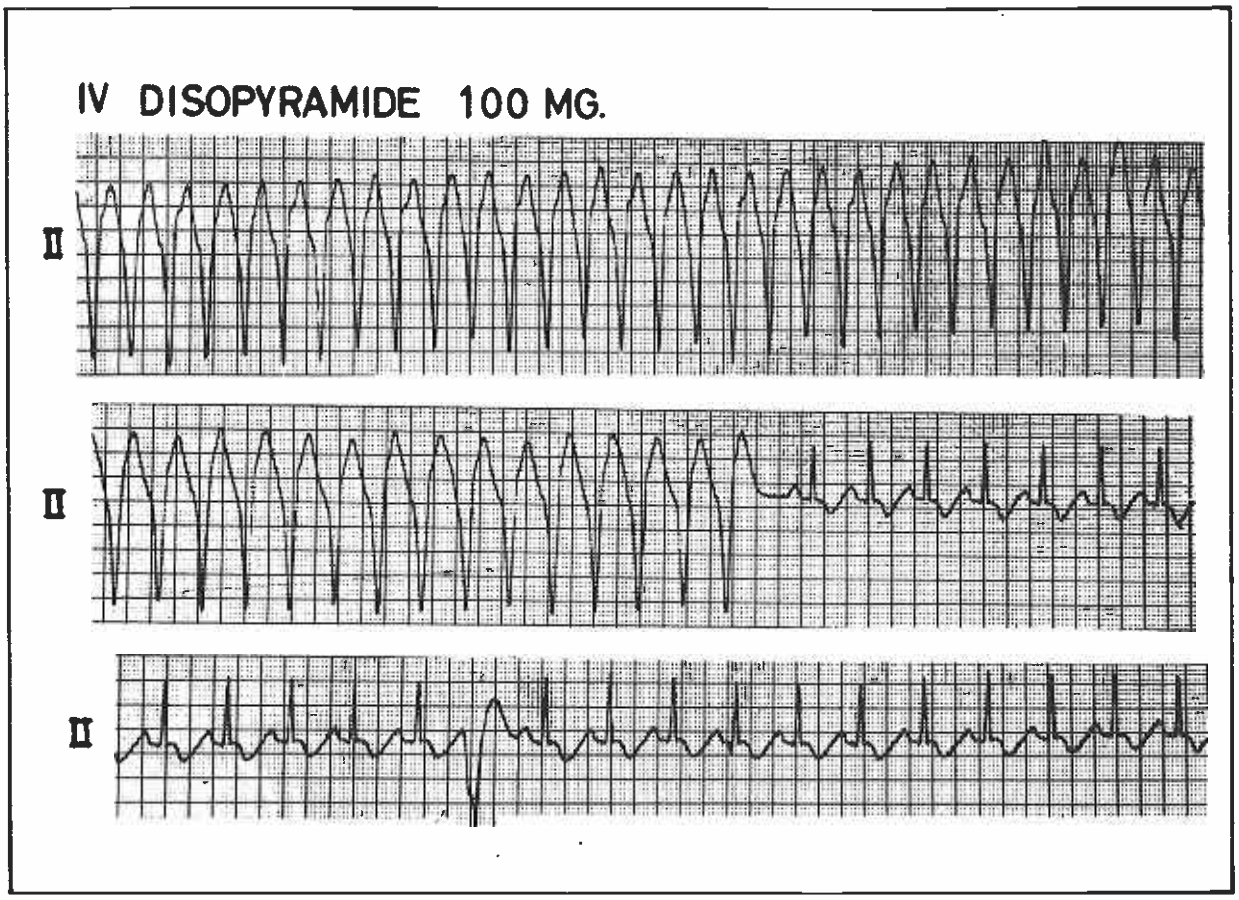

Fig. 1 Termination of ventricular tachycardia following i.v. disopyramide. (ECG strips are continuous).

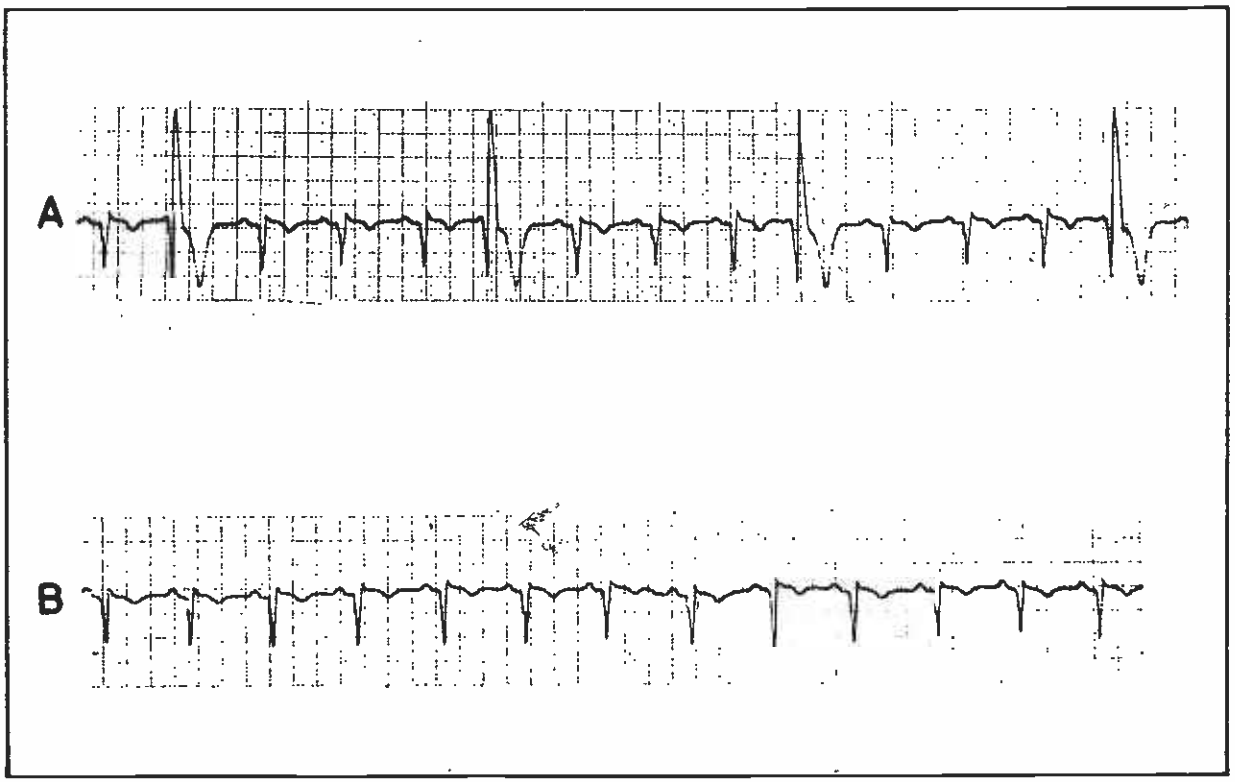

Fig. 2 Complete suppression of frequent ventricular ectopic beats following I.V. disopyramide. (Panel A was recorded before and Panel B after i.v. disopyramide).

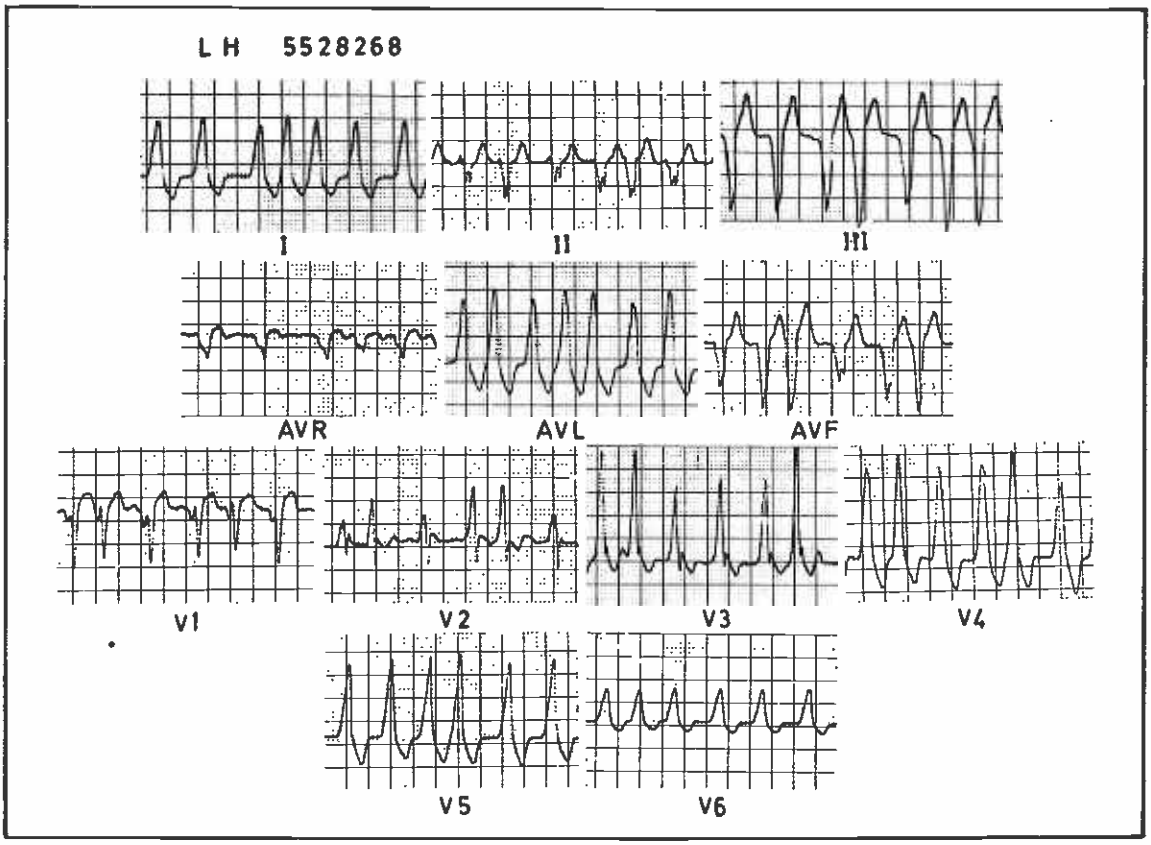

Fig. 3 ECG of patient with rapid atrial fibrillation and WPW conduction abnormality.

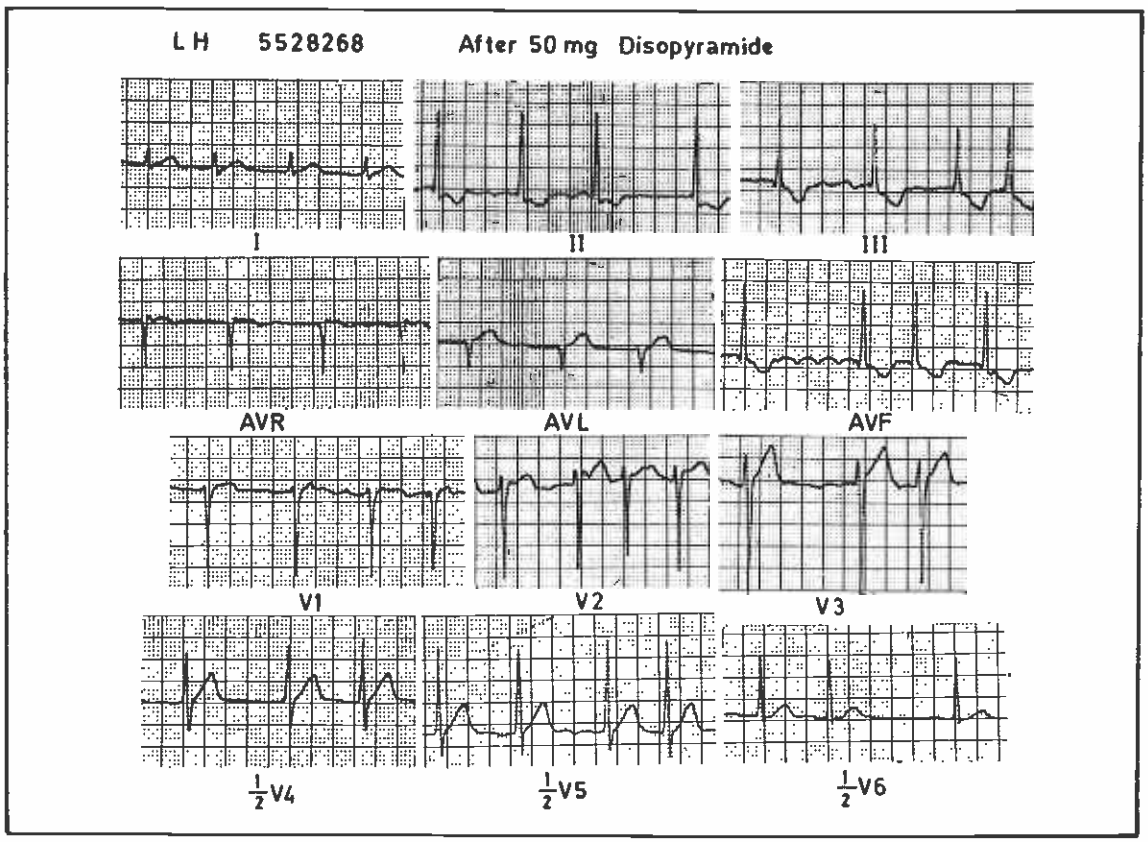

Fig. 4 ECG of same patient as in Fig. 3 after 50 mg i.v. disopyramide showing atrial fibrillation but normal atrioventricular conduction.

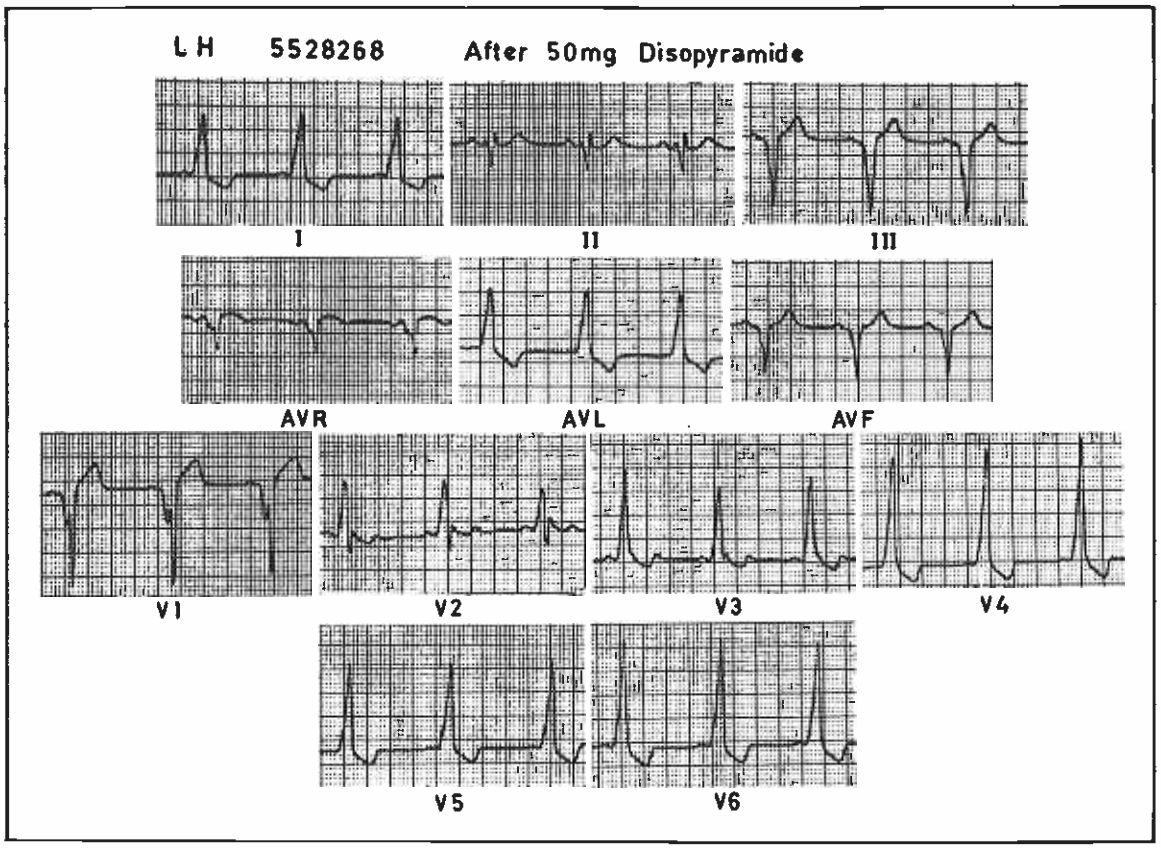

Fig. 5 ECG recorded soon after Fig. 4 showing sinus rhythm and WPW conduction abnormality.

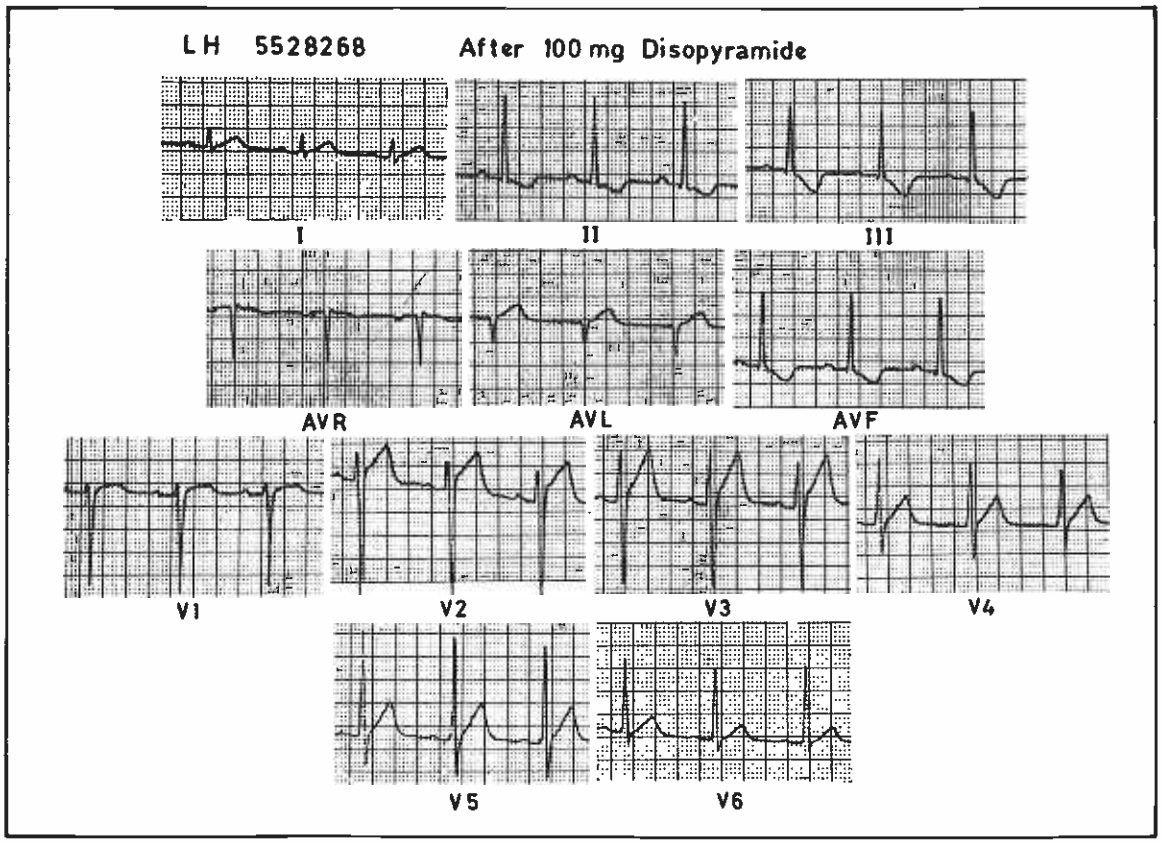

Fig. 6 ECG recorded after administration of 100 mg i.v. disopyramide showing sinus rhythm and normal conduction.

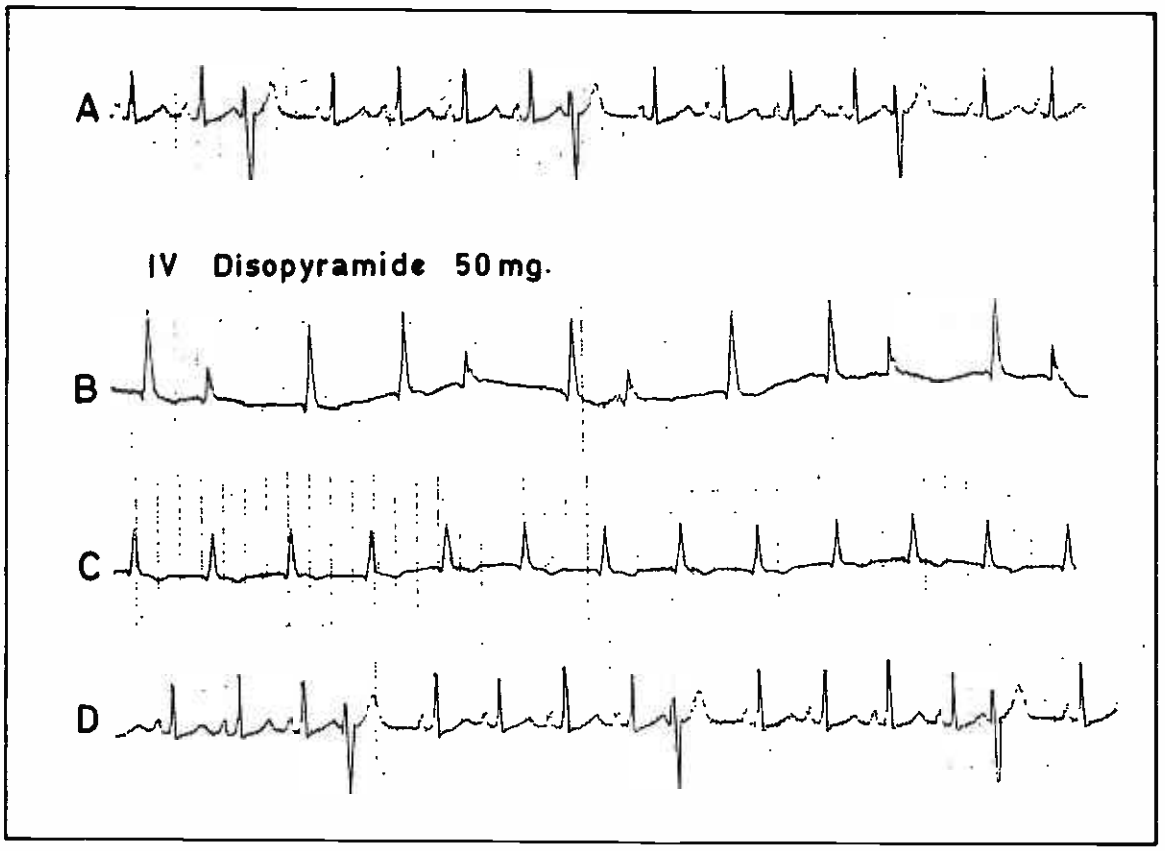

Fig. 7 Panel A shows ECG before i.v. disopyramide in a 55 year old man with recent anterior infarction and heart failure. Panel B was recorded after 50 mg of i.v. disopyramide. Panel C was recorded soon after and Panel D about 2 hours following an intravenous isoprenaline infusion.

success rate of intravenous disopyramide was even higher (7). They reported successful termination in 57% of patients with atrial fibrillation, 83% with supraventricular tachycardia, 88% with ventricular tachycardia and suppression of ventricular ectopic beats in 86% of patients. Unlike the favourable results of Deano et al but in accordance with the findings of Mizgala et al (6), Tonkin and co-workers found that intravenous disopyramide terminated atrial flutter in only 12% of cases (7).

Our present study confirms previous findings that intravenous disopyramide is a very effective antiarrhythmic agent when used for the purpose of terminating ventricular tachycardia, atrial tachycardia and for the suppression of ventricular and atrial ectopic beats. In our experience the success rate for suppression of ventricular tachycardia was 75%, for ventricular ectopic beats 86%, for atrial ectopic beats and atrial tachycardia 100% and for normalizing WPW conduction 100%. Our study also supports the findings of Bennett who reported that intravenous disopyramide is useful for slowing the ventricular rate and normalizing the WPW conduction abnormality in such patients presenting with atrial fibrillation (8). Like Tonkin et al and Mizgala et al, we found intravenous disopyramide to be ineffective in atrial flutter. Therefore the use of this drug in this condition appears to be unwarranted. One of the advantages of using intra venous disopyramide for the acute termination of cardiac arrhytmias is that oral disopyramide can be started and continued as maintenance therapy immediately after a successful intravenous injection.

In this study, the side effects of intravenous disopyramide were by and large minimal. A transient but clinically insignificant fall in systolic pressure of less than 20 mmHg was seen in 5 out of our 39 patients. However in 1 patient with heart failure, intravenous disopyramide produced severe bradycardia due to a junctional rhythm and severe hypotension both of which responded to an isoprenaline infusion. Therefore in patients with cardiac failure or shock, intravenous disopyramide is contraindicated, and if it has to be given, must be administered with the greatest of care.

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