Brugada syndrome is a clinical and electrocardiographic entity characterized by ST segment elevation in the right precordial ECG leads and sudden death or syncope secondary to malignant ventricular arrhythmia, and has a high recurrence rate. We report a patient with this syndrome who had received an automatic implantable defibrillator, who presented with multiple appropriate discharges because of recurrent episodes of ventricular fibrillation. All episodes were started by a premature ventricular beat of the same morphology and coupling interval. Endovenous procainamide administration, paradoxically, was effective in preventing new episodes. The beneficial antiarrhythmic effect of procainamide in this patient is discussed.

**Key words:** Brugada syndrome. Ventricular fibrillation. Drugs.

**INTRODUCTION**

Brugada syndrome is a clinical and electrocardiographic entity characterized by a typical ECG pattern and syncope or sudden death.¹ The condition involves malignant ventricular arrhythmia, polymorphic ventricular tachycardia and ventricular fibrillation and has a high recurrence rate.² The episodes usually present alone, but occasionally appear as multiple episodes of arrhythmia. At present there is little information on the origin and treatment of this condition, although several potential triggering factors have been suggested recently.³ We describe a patient with this syndrome who had received an implantable cardioverter defibrillator (ICD) and presented arrhythmia with multiple appropriate discharges secondary to episodes of recurrent ventricular fibrillation.

**CASE STUDY**

A 54-year-old man was seen in April 1996 after aborted sudden death secondary to ventricular fibrillation. The patient had no personal or family history of interest and the physical examination was normal. The electrocardiogram revealed the characteristic pattern of Brugada syndrome (Figure 1). The echocardiogram and cardiac catheterization studies were normal. Electrophysiological study showed an HV interval of 60 ms, and ventricular fibrillation was induced by a drive train followed by two ventricular extrastimuli,
with coupling of 600/250-180 ms from the apex of the right ventricle. The administration of 20 mg of ajmaline produced marked elevation of the J point and ST segment, which normalized with isoproterenol administration (Figure 1). A single-chamber implantable cardioverter defibrillator was implanted.

On two occasions (August 1998 and November 2001), the patient presented at the emergency room
sient outward potassium current (Ito) has been suggested, this effect has not been shown in the case of procainamide. Nevertheless, chronic administration of oral procainamide was not used to prevent recurrence, because of the high incidence of medium- and long-term adverse effects associated with this type of regimen.

We observed that all documented episodes in our patient started with a premature ventricular beat of the same morphology and similar coupling in the morphology channel of the implantable cardioverter defibrillator. The role of premature ventricular beats in triggering ventricular fibrillation, as well as its treatment with catheter ablation, have recently been reported.5,6 We attribute the success of procainamide in our patient to suppression of the premature beats, rather than to an effect on the actual mechanism of the syndrome.

DISCUSSION

The frequency of Brugada syndrome has not been estimated, although a number of cases of have been reported in the literature.3,4 There is little information about arrhythmias in patients with this condition. Although several potential triggering factors such as autonomic tone, temperature3 or the use of antiarrhythmic drugs have been suggested, none were found in our patient.

Treatment has not been established as yet, although isoproterenol has been reported to be effective.4 This drug was used in our patient because its therapeutic usefulness was not known at the time of the first episode (1998) and because the patient responded well to procainamide administration in the second (2001).

In view of the underlying abnormality of this disorder, it is paradoxical that procainamide was successful in our patient. Although the therapeutic benefit of quinidine at low doses caused by blockade of the tran-

REFERENCES