

# Epicardial Electrogram of the Right Ventricular Outflow Tract in Patients With the Brugada Syndrome

## Using the Epicardial Lead

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<b>OBJECTIVES</b>	We tried to record an epicardial electrogram directly, and we examined local electrograms before and after administration of a class IC anti-arrhythmic drug in patients with the Brugada syndrome.
<b>BACKGROUND</b>	Electrical heterogeneity of the epicardium in the right ventricular outflow tract (RVOT) has been thought to be related to the Brugada syndrome. However, an epicardial abnormality has not been demonstrated in patients with the Brugada syndrome.
<b>METHODS</b>	In five patients with a Brugada-type electrocardiogram (ECG), local unipolar electrograms were recorded at the epicardium and endocardium of the RVOT. To record the epicardial electrogram directly, we introduced an electrical guidewire into the conus branch (CB) of the right coronary artery. The duration of the local electrogram after termination of the QRS complex (DP) was measured before and after class IC anti-arrhythmic drug administration. The signal-averaged electrocardiogram (SAECG) was also obtained in all patients.
<b>RESULTS</b>	A definite DP was observed at the epicardium, but not at the endocardium. After administration of a class IC anti-arrhythmic drug, the DP at the epicardium was prolonged from $38 \pm 10$ ms to $67 \pm 24$ ms. The late potential corresponding to the DP at the epicardium was observed in all patients on the SAECG.
<b>CONCLUSIONS</b>	An epicardial electrogram can be recorded from the CB. Recording from the CB enables identification of an epicardial abnormality in patients with the Brugada syndrome. These abnormal electrograms may be related to a myocardial abnormality in the epicardium of patients with the Brugada syndrome. (J Am Coll Cardiol 2002;39:1992-5) © 2002 by the American College of Cardiology Foundation

The Brugada syndrome is an idiopathic ventricular fibrillation (VF) characterized by ST-segment elevation in the precordial leads, and is augmented by sodium channel blockers (1-3). It has been suggested that electrical heterogeneity, especially at the epicardium of the right ventricle, causes the ST-segment elevation and is related to the occurrence of polymorphic ventricular arrhythmias (4,5).

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However, studies using body surface mapping or the signal-averaged electrocardiogram (SAECG) have suggested that a conduction delay exists in the anterior wall of the right ventricle or the right ventricular outflow tract (RVOT) in patients with the Brugada syndrome (6,7). However, endocardial mapping in a routine electrophysiologic study failed to demonstrate this electrical abnormality.

The conus branch (CB) arises at the right coronary ostium or within the first few millimeters of the right coronary artery. It runs on the surface of the free wall of the

right ventricular outflow tract (RVOT-FW). Therefore, an electrogram obtained at the CB may reflect an epicardial electrogram at the RVOT-FW. In this study, we analyzed electrograms recorded at the CB and examined the effects of class IC anti-arrhythmic drugs in patients with the Brugada syndrome.

## METHODS

**Study group.** Five male patients (age  $45 \pm 11$  years) with the Brugada syndrome were studied. All patients had persistent ST-segment elevation in the precordial leads. In three patients, episodes of VF or aborted sudden cardiac death were observed (i.e., symptomatic patients). In the other two patients, no episodes of VF or aborted sudden cardiac death were observed, but VF was demonstrated with single or double extrastimulation at the RVOT-FW in the electrophysiologic study (i.e., asymptomatic patients). Routine studies, including cardiac echocardiography, coronary angiography, right and left ventriculography, radionuclide angiography and endomyocardial biopsy, showed no evidence of structural heart disease.

**Electrophysiologic study.** After obtaining written, informed consent, an electrophysiologic study was performed in all patients.

After right coronary arteriography, an electrical guidewire (FloWire, Cardiometrics, California) was introduced into

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**Abbreviations and Acronyms**

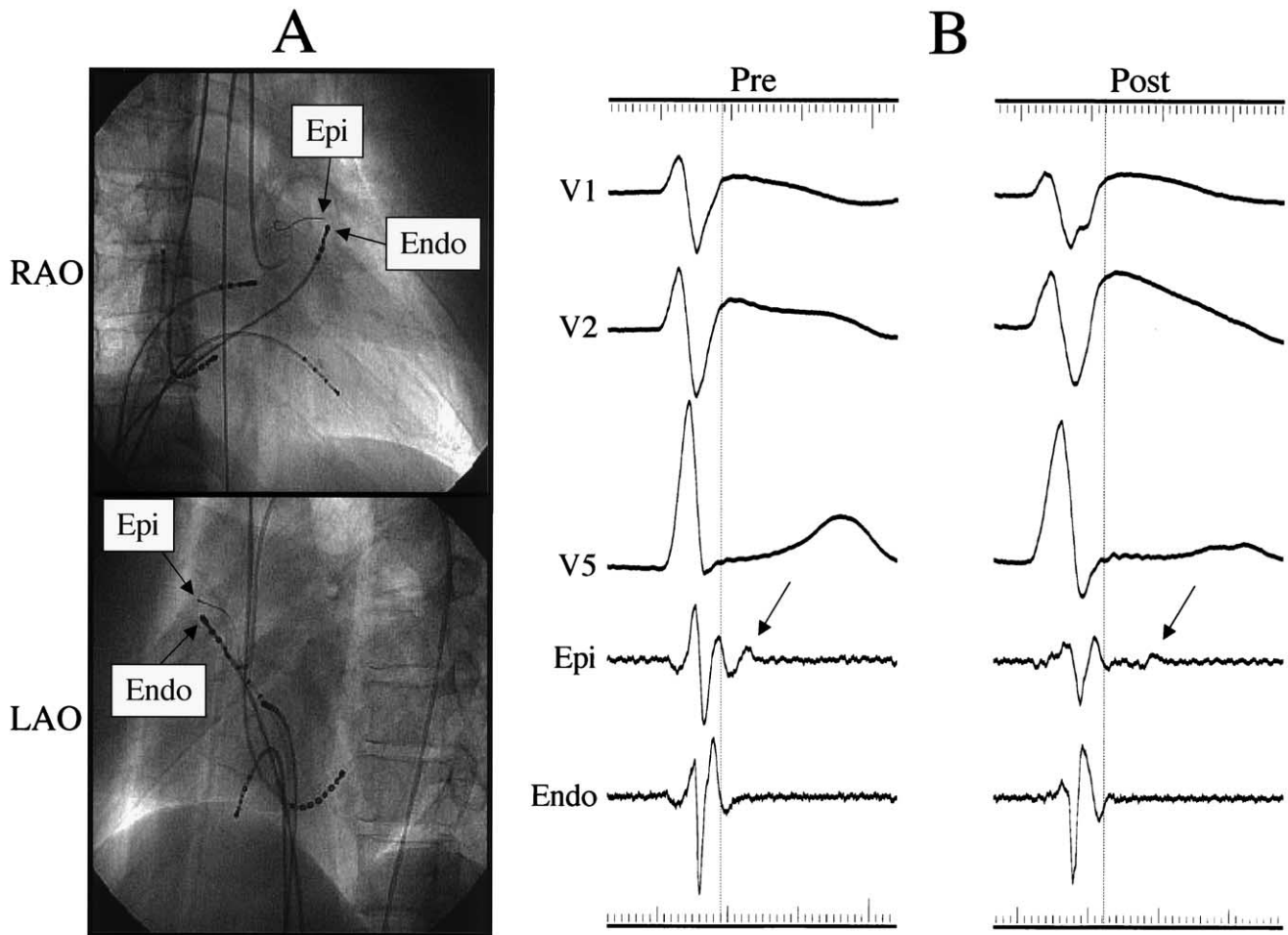
CB	= conus branch
DP	= local electrogram after termination of the QRS complex
ECG	= electrocardiogram/electrocardiographic
LAS <sub>40</sub>	= duration of low-amplitude signals <40 $\mu$ V in the terminal filtered QRS complex
LP	= late potential
RMS <sub>40</sub>	= root mean square voltage of the terminal 40 ms in the filtered QRS complex
RVOT	= right ventricular outflow tract
RVOT-FW	= free wall of the right ventricular outflow tract
SAECG	= signal-averaged electrocardiogram
VF	= ventricular fibrillation

the CB through the right coronary artery. A local unipolar electrogram at the epicardium of the RVOT-FW was recorded at the tip of the wire, using a 30- to 400-Hz

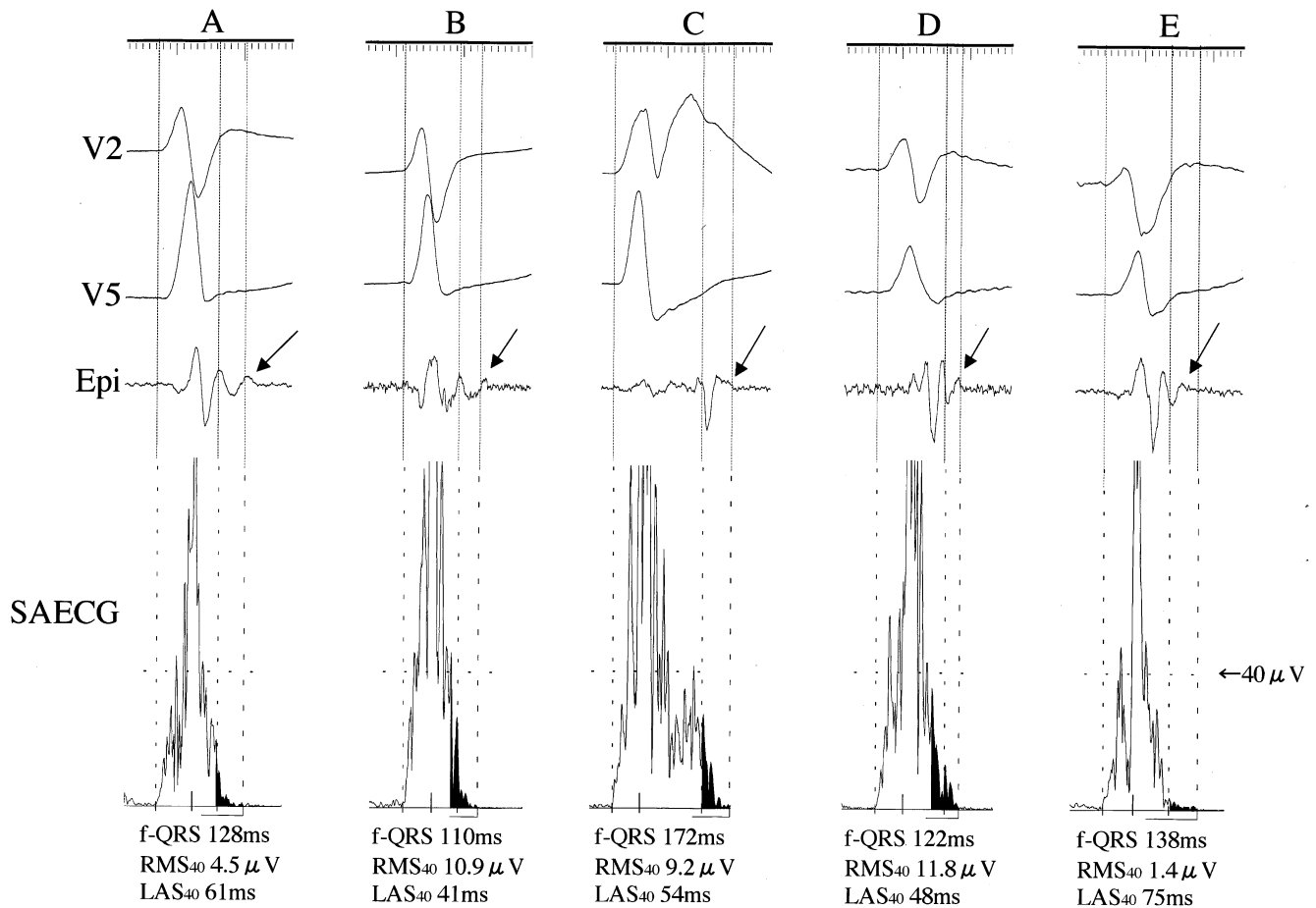
bandwidth. A local unipolar electrogram at the endocardium was recorded using a 30- to 400-Hz bandwidth, by a quadripolar 6F deflectable catheter positioned at the endocardium at the RVOT-FW (Fig. 1A).

The duration of the local electrogram after termination of the QRS complex (DP) was measured before and after class IC anti-arrhythmic drug administration. Termination of the QRS complex was defined in lead V<sub>5</sub>. Pilsicainide (0.7 to 1.0 mg/kg) was administered to Patients 1 to 4, and flecainide (1.0 mg/kg) was administered to Patient 5 as a bolus over a period of 10 min.

**SAECG.** The SAECG (ART 1200EPX; noise level <0.3  $\mu$ V, high-pass filtering of 40 Hz using a bidirectional four-pole Butterworth) was performed in all patients. The filtered QRS duration, the root mean square voltage of the terminal 40 ms in the filtered QRS complex (RMS<sub>40</sub>) and the duration of low-amplitude signals <40  $\mu$ V in the terminal filtered QRS complex (LAS<sub>40</sub>), were measured



**Figure 1.** (A) Fluoroscopic 30° right anterior oblique (RAO) and 60° left anterior oblique (LAO) views of the position of the electrical guidewire introduced into the conus branch through the right coronary artery for epicardial mapping (Epi), as well as the quadripolar deflectable catheter at the endocardium of the free wall at the right ventricular outflow tract for endocardial mapping (Endo). (B) Surface electrocardiographic leads V<sub>1</sub>, V<sub>2</sub> and V<sub>5</sub> and intracardiac electrograms of the epicardium (Epi) and endocardium (Endo) at the free wall at the right ventricular outflow tract before (Pre) and after (Post) the administration of a class IC anti-arrhythmic drug in Patient 1 (asymptomatic patient). A definite local electrogram was recorded after termination of the QRS complex at the epicardium (arrows), and the duration of the local electrogram after termination of the QRS complex after class IC anti-arrhythmic drug administration was more prolonged at the epicardium than at the endocardium in Patient 1.



**Figure 2.** (A to E) Surface electrocardiographic leads V<sub>2</sub> and V<sub>5</sub>, intracardiac electrograms of the epicardium (Epi) at the free wall at the right ventricular outflow tract and signal-averaged electrocardiogram (SAECG) in Patients 1 (A), 2 (B), 3 (C), 4 (D) and 5 (E). Patients 1 and 2 are asymptomatic patients, and Patients 3, 4 and 5 are symptomatic patients. The late potentials, represented by the shaded area, are positive in all patients, showing the root mean square voltage of the terminal 40 ms in the filtered QRS complex (RMS<sub>40</sub>) <20  $\mu$ V and the duration of low-amplitude signals <40  $\mu$ V in the terminal filtered QRS complex (LAS<sub>40</sub>) >40 ms, and correspond to a delayed potential after the QRS complex at the epicardium (arrows) in all patients. f-QRS = filtered QRS duration.

with SAECG. A late potential (LP) was considered to be positive when two criteria (RMS<sub>40</sub> <20  $\mu$ V and LAS<sub>40</sub> >40 ms) were met.

## RESULTS

A definite DP was observed at the epicardium, but not at the endocardium. After administration of a class IC antiarrhythmic drug, the duration of the DP at the epicardium was prolonged from  $38 \pm 10$  ms to  $67 \pm 24$  ms (Fig. 1B). An LP corresponding to the DP at the epicardium was observed in all patients on the SAECG (Fig. 2).

## DISCUSSION

The Brugada syndrome is an idiopathic VF of uncertain etiology (1-3). Several studies have suggested that electrical heterogeneity is related to the genesis of the Brugada syndrome (4,5). A depressed right ventricular epicardial action potential dome is the basis for the ST-segment elevation and leads to phase 2 re-entry as a trigger for

episodes of polymorphic ventricular arrhythmias. However, the existence of a conduction delay at the RVOT has been suggested in many reports (6,7). In addition, several clinical studies have suggested that spontaneous episodes of VF are triggered by premature ventricular contractions originating from the right ventricular free wall (8,9). However, this electrical abnormality was not demonstrated by endocardial mapping in a routine electrophysiologic study.

In patients with the Brugada syndrome, class IC antiarrhythmic drug administration could augment ST-segment elevation in the precordial leads, and spontaneous VF has sometimes been observed (3,10).

The CB arises at the right coronary ostium or within the first few millimeters of the right coronary artery. It runs on the surface of the RVOT-FW. In this study, we were able to directly record a unipolar epicardial electrogram at the RVOT-FW, by using an electrical guidewire introduced into the CB. There was a delayed potential at the QRS complex in the epicardium of the RVOT-FW, which was not demonstrated by endocardial mapping. Class IC anti-

arrhythmic drug administration augmented ST-segment elevation and also augmented the DP in the epicardium. An LP was observed on the SAECG in all patients and corresponded to the DP recorded on the epicardial electrogram. These abnormal electrograms were thought to reflect a myocardial abnormality in the epicardium.

Because the CB is attached to the left ventricular cavity through the low resistance of blood, it is quite possible that the DP recorded in the CB reflects not only local electrograms at the RVOT, but also remote regions such as the left ventricle. To exclude that possibility, left ventricular endocardial mapping was performed in all patients, and the DP was not demonstrated at the left ventricular endocardium in any of the patients. Furthermore, left ventricular epicardial electrograms of the great cardiac vein through the coronary sinus were recorded in some patients who did not show a DP.

**Conclusions.** Recording from the CB enables identification of an epicardial abnormality in patients with the Brugada syndrome. Class IC anti-arrhythmic drug administration worsens these abnormalities, which may play an important role in the occurrence of VF in patients with the Brugada syndrome.

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