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IMAGES IN ELECTROPHYSIOLOGY

Catheter Ablation of Brugada Syndrome

Importance of Repeated Administration of Ajmaline to Unmask the Entire Epicardial Substrate

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A 51-year-old man with Brugada syndrome (BrS) was admitted for implantable cardioverter-defibrillator implantation following out-of-hospital cardiac arrest due to ventricular fibrillation. Given the dramatic event and high risk of ventricular fibrillation recurrence, the previously asymptomatic patient asked about available treatment options. Antiarrhythmic drug therapy (quinidine) and epicardial catheter ablation were discussed with the patient, and he opted for an interventional approach.

After implantable cardioverter-defibrillator implantation, the patient underwent epicardial mapping of the right ventricle (RV). Using an anterior epicardial access and a 3-dimensional mapping system (NavX, St. Jude Medical, St. Paul, Minnesota) with a linear multipolar mapping catheter, we performed voltage, activation, and potential duration mapping of the RV epicardium. The baseline map (**Figure 1A**) revealed a small area with fractionated and prolonged potentials (duration >110 ms measured from QRS onset) over the RV outflow tract (RVOT). Interestingly, after ajmaline administration (30 mg) and the induction of coved-type electrocardiogram (ECG) in right precordial leads (**Figure 1B**), these potentials expanded both in duration and distribution extending down to the RV free wall (**Figure 1B**). Fractionated potentials with prolonged duration beyond the QRS end were targeted by ablation (30 W, irrigated radiofrequency) leading to further dramatic increase of ST-segment elevation during ablation (**Figure 1C**). Afterwards, another ajmaline administration (30 mg) induced coved-type ECG only in lead V_1 (**Figure 1D**) (V_1 placed in second intercostal space over the RVOT) corresponding to residual prolonged and fractionated electrograms in the RVOT (**Figure 1D**). After reablation of these potentials, a final remap with ajmaline (30 mg) demonstrated a normal potential duration in the entire RV epicardium and only horizontal or ascending ST-segment elevation (**Figure 1E**).

Since the seminal study by Nademanee et al. (1) describing abnormal electrograms over the RVOT epicardium and successful prevention of ventricular fibrillation recurrence by catheter ablation of these potentials in BrS patients, Sacher et al. (2) and Brugada et al. (3) demonstrated that sodium-channel blockade can prolong the duration of abnormal epicardial electrograms and reveal an arrhythmogenic substrate extending beyond the RVOT. To our knowledge, we present one of the first illustrations of: 1) re-induction of coved-type ECG by ajmaline after epicardial ablation; caused by 2) residual abnormal electrograms in the corresponding epicardial RVOT region; and 3) disappearance of coved-type ECG after abolition of the remaining epicardial substrate. To achieve complete

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All authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Clinical Electrophysiology* author instructions page.

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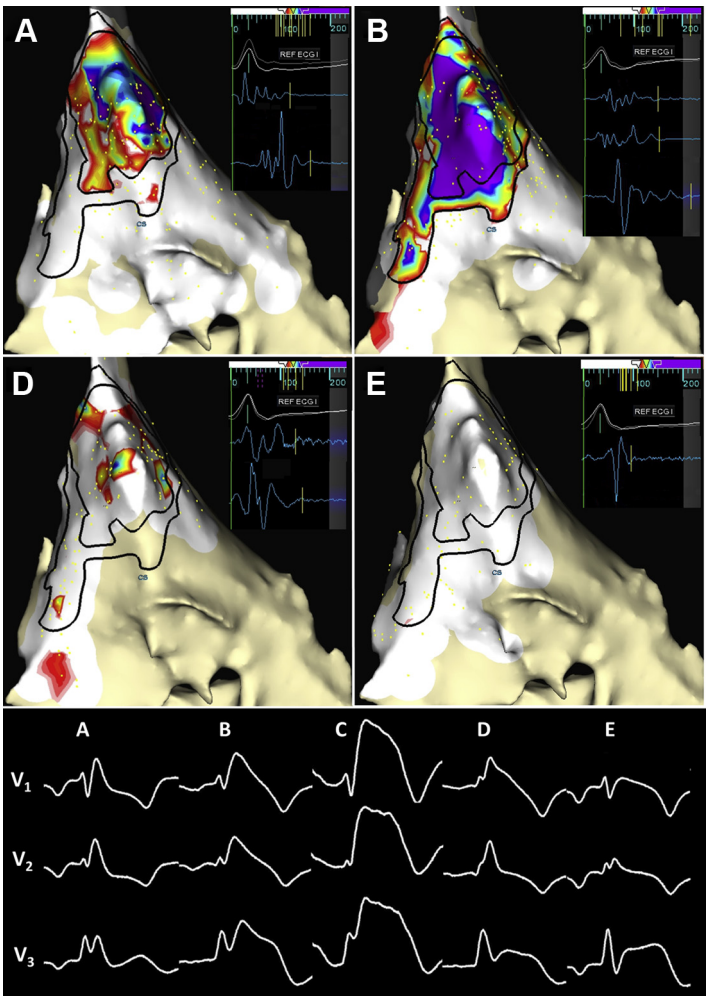
FIGURE 1 Epicardial RV Potential Duration Maps (AP View), Corresponding Epicardial EGMs and Right Precordial Leads



(A) Baseline, **(B)** after ajmaline, **(C)** during epicardial radiofrequency catheter ablation of fractionated and prolonged potentials, **(D)** ajmaline remap after first epicardial ablation, and **(E)** final ajmaline remap after second epicardial ablation. AP = anterior posterior; ECG = electrocardiogram; EGMs = electrograms; RV = right ventricle.

substrate elimination, catheter ablation in BrS should be guided by repeated administration of ajmaline to unmask the entire epicardial substrate. This substrate-based, interventional approach could pave the way to a cure for BrS. Our observations are in line with the depolarization disorder hypothesis as the main electrophysiological mechanism underlying BrS (4).

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