Flecainide Enhances Endocardial Unipolar Voltage Abnormalities in Brugada Syndrome

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A 40-year-old male patient with Brugada syndrome underwent high-density endocardial voltage mapping (>1000 points) of the right ventricular outflow tract (RVOT) using a three-dimensional mapping system. Electroanatomical maps were performed before and after flecainide infusion (70 mg), setting the reference value for normal unipolar electrograms at >4 mV. A broader area of abnormal unipolar signals was developed at the free wall of the RVOT after flecainide infusion. Endocardial unipolar mapping after sodium channel challenge may accurately identify the epicardial arrhythmogenic substrate and may have potential clinical implications in endocardial ablation strategies.

Brugada syndrome (BrS) is a genetic arrhythmia syndrome with increased risk of sudden cardiac death. Unmasking a type I BrS electrocardiogram (ECG) pattern after administration of sodium channel blockers may be associated with an increase in epicardial substrate abnormalities. We hypothesised that high-density endocardial unipolar voltage mapping of the right ventricular outflow tract (RVOT), before and after flecainide infusion in a patient with BrS, may detect enhancement of the electroanatomical abnormalities possibly related to the modification of the abnormal epicardial tissue.

A 40-year-old male asymptomatic patient with BrS was referred to our centre and underwent electrophysiological study for risk stratification via programmed ventricular stimulation. Initially, he underwent high-density endocardial voltage mapping of the RVOT during sinus rhythm. Cardiac-magnetic resonance imaging ruled out structural heart disease. More than 1,000 points were sampled throughout the RVOT using a multi-electrode mapping catheter. The reference value for normal unipolar electrograms was set at >4 mV and electroanatomical maps were performed before and after flecainide infusion (70 mg). As shown in Figure 1, the patient displayed a small area of unipolar voltage abnormalities beneath the pulmonary valve (Figure 1A). After administration of flecainide, a Brugada ECG pattern with typical coved ST elevation >2 mm in right precordial leads is seen (Figure 1B), and a broader area of abnormal unipolar signals was developed at the free wall of the RVOT (Figure 1B). Programmed ventricular stimulation was performed from the right ventricle, inducing ventricular fibrillation (VF) (Figure 3). Endocardial unipolar mapping after sodium channel challenge may accurately identify the epicardial arrhythmogenic substrate and may have potential clinical implications in endocardial ablation strategies.

The endocardial unipolar voltage abnormalities possibly reflect the epicardial bipolar voltage abnormalities that have been detected at the RVOT of patients with BrS. Venlet et al. have shown that the optimal endocardial unipolar voltage cut-off for the identification of epicardial right ventricular scar was 3.9 mV in patients who underwent endocardial and epicardial right ventricular voltage mapping.2 Venlet et al. have elegantly demonstrated a significant increase in low bipolar voltage epicardial areas in RVOT after flecainide infusion.2 Flecainide facilitates the identification of the extension and distribution of arrhythmia substrate during epicardial mapping.2 Epicardial substrate elimination was associated with normalisation of BrS ECG pattern and the absence of ventricular tachycardia/VF inducibility in addition, an endocardial ablation approach has been shown to normalise the ECG and to supress VF storms.2 Endocardial unipolar mapping after sodium channel challenge may accurately identify the epicardial arrhythmogenic substrate and may have potential clinical implications in endocardial ablation strategies.
A broader area of abnormal unipolar electrograms was developed at the free wall of the RVOT after 70 mg of flecainide infusion (A). Similarly, the multipolar catheter DecaNav® (Biosense-Webster Inc., Irvine, CA, US) recorded abnormal multicomponent bipolar signals after drug challenge (B). The ECG (lead V1, positioned at the third ICs) revealed a coved type ST segment elevation. DECA = DecaNAV; ECG = electrocardiogram; RVOT = right ventricular outflow tract.

Figure 2: Electrocardiographic changes after flecainide infusion

Precordial leads were positioned at the second (V1–V2), third (V3–V4) and fourth (V5–V6) ICs. A coved ST–T segment elevation >2 mm was unmasked after 70 mg of flecainide infusion. ICs = intercostal space.

Programmed ventricular stimulation was performed from the right ventricle, inducing ventricular fibrillation at drive train with a 500 ms cycle length and two extra stimuli (500–230–250 ms).