

Editorial

## Ventricular Arrhythmias Ablation in Brugada Syndrome. Current and Future Directions

### Ablación de arritmias ventriculares en el síndrome de Brugada. Presente y futuro

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Brugada Syndrome (BrS) is characterized by an elevated ST segment in the right precordial leads (V<sub>1-3</sub>) on the electrocardiogram (ECG) and an increased risk of ventricular tachycardia/ventricular fibrillation (VT/VF) episodes and sudden cardiac death.<sup>1,2</sup> Although more than 2 decades have passed since BrS was first reported by the Brugada brothers,<sup>1</sup> few therapeutic options have been found, despite the strong interest in this syndrome. Indeed, at present, there are just 2 therapeutic strategies, which include implantable cardioverter-defibrillator (ICD) and/or chronic quinidine therapy.<sup>2</sup> However, quinidine is not effective in many patients and its use is frequently associated with intolerable adverse effects. ICD implantation may be effective in preventing sudden cardiac deaths, and is currently recommended as a class I indication for symptomatic patients with type 1 Brugada ECG pattern who present with aborted sudden death and VF-related symptoms such as syncope, seizure, or nocturnal agonal respiration. Unfortunately, ICD therapy in many patients is associated with inappropriate shocks, lead fractures/failure, and device infections. Additionally, high-risk patients with BrS have recurrent VF episodes, which cause frequent ICD discharges or storms. Rarely, heart transplantation is necessary to control electrical storm.

#### THE DISCOVERY AND CHARACTERIZATION OF THE ARRHYTHMIC SUBSTRATE IN BRUGADA SYNDROME

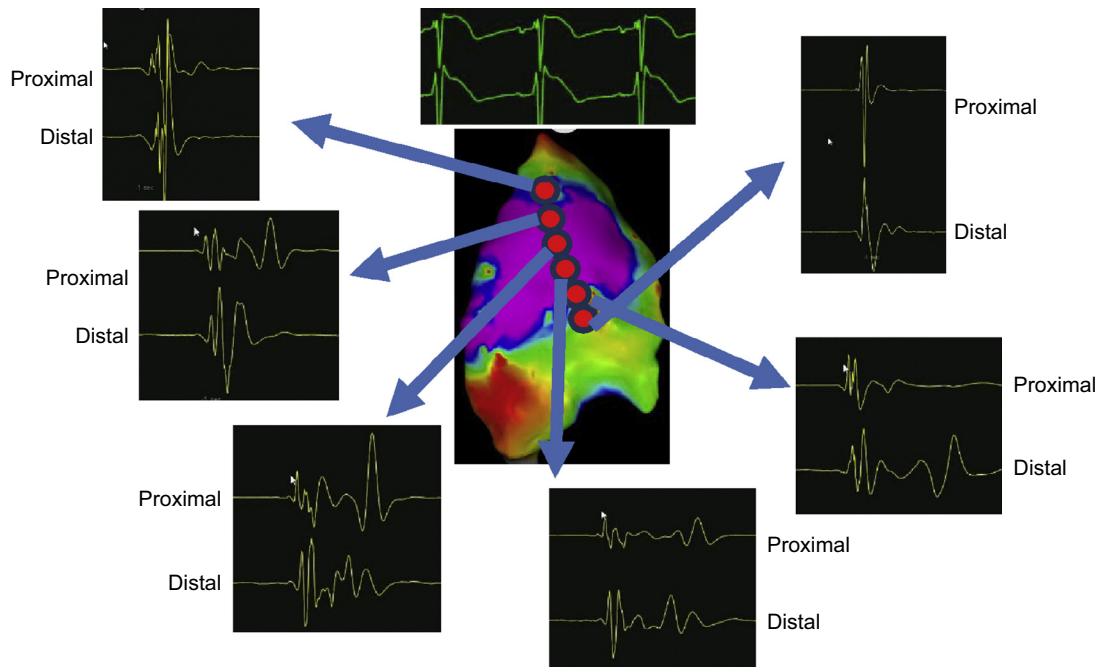
Past mapping and ablation techniques, initially performed in a limited number of selected BrS patients, targeted both epicardium and endocardium with unsatisfactory results. Currently, there is consensus that potentially arrhythmic substrates responsible for the abnormalities seen in the typical BrS ECG pattern are located on the anterior right ventricular outflow tract of the pericardium.<sup>3-10</sup> Lacking effective and well-defined substrate-based therapeutic strategies to control and prevent malignant ventricular tachyarrhythmias in BrS, we first attempted to identify any potential arrhythmic substrates among a large BrS population with different clinical presentations and different baseline BrS ECG patterns.<sup>4,6</sup> We did so using combined endo- and epicardial mapping using 3-dimensional potential duration maps before and after ajmaline

infusion (1 mg/kg in 5 minutes). Extensive electrophysiologically well-defined abnormal areas, as unmasked by ajmaline, were accurately identified exclusively on the anterior right ventricular outflow tract and/or right ventricular anterior free wall of the pericardium<sup>6</sup> (Figure 1). Wider abnormal areas were found in patients with the worst clinical presentation and/or type 1 BrS ECG pattern. However, in less symptomatic patients, similar large abnormal areas were unmasked after ajmaline infusion.<sup>6</sup> In less symptomatic patients without typical BrS-related symptoms, the substrate increased more than 3 times after ajmaline, suggesting that in such a patient population the presence or persistence of modulating or aggravating factors is required to activate the substrate in order to facilitate the development of VT/VF. Overall, the ajmaline-induced substrate increase was larger in men than in women,<sup>6</sup> confirming previous studies on a sex difference in BrS. Of interest, low-voltage (< 1 mV) fragmented very prolonged potentials were found exclusively in patients with the worst clinical presentation, while in less symptomatic BrS patients we found abnormal ventricular potentials with preserved voltage amplitude.<sup>6</sup> While demonstrating the role of extensive complex epicardial substrates, our substrate-based strategy clearly explains the ineffectiveness of endocardial ablation alone, as well as the lower success of less extensive epicardial ablation not systematically guided by Class Ic drug administration, as initially proposed by others. In our experience, ajmaline rechallenge after ablation can reveal additional abnormal potentials in many patients (> 60%), requiring further RF applications to persistently normalize the ECG pattern.<sup>6</sup> Only after ablation of all residual substrates, as confirmed by ajmaline rechallenge, can we persistently normalize the ECG pattern rendering VT/VF noninducible<sup>6</sup> (Figure 2). These data and the role of ajmaline infusion are indeed important for successful electrical substrate ablation in BrS. As a result, for the first time, substrate-based ablation strategy, as proposed by our group, could now be available for the vast majority of patients with BrS.

#### AJMALINE INFUSION AS A NOVEL METHOD TO UNMASK AND DETERMINE THE BRUGADA SYNDROME SUBSTRATE

We proposed for the first time ajmaline infusion as a novel method to accurately determine the location and size of arrhythmic electrical substrate (AES) in a series of 14 patients.<sup>4</sup>

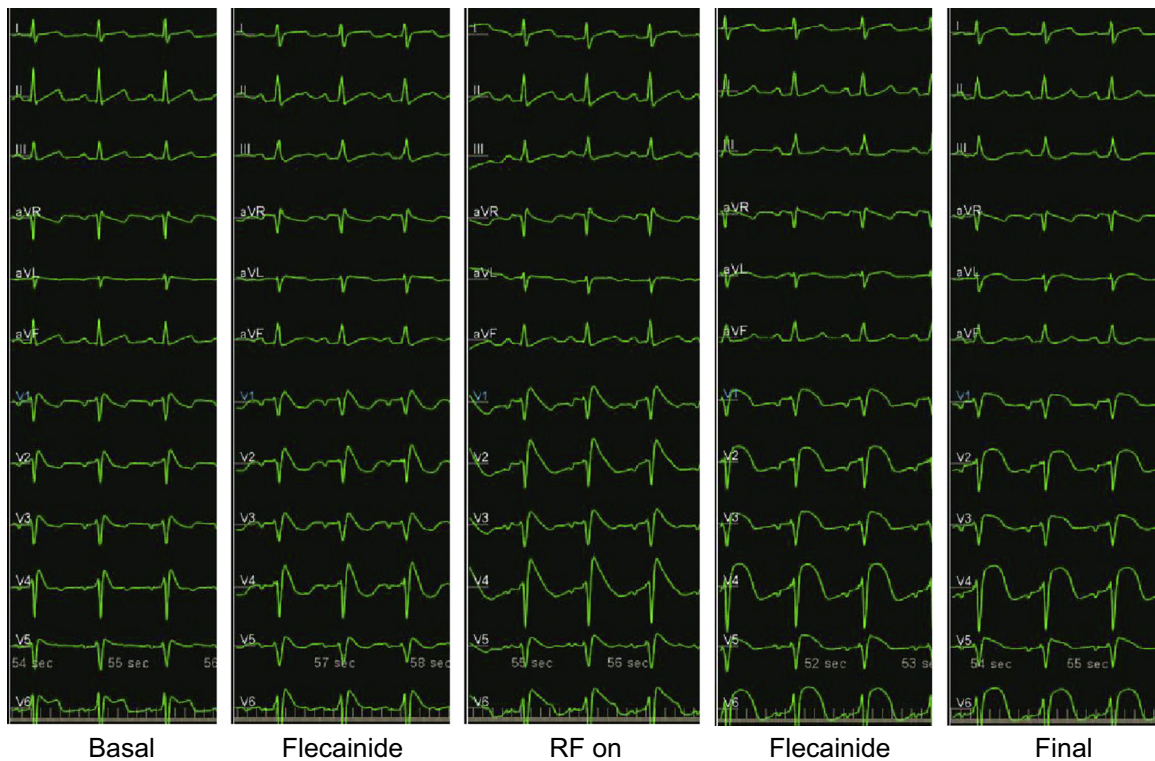
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**Figure 1.** Different samples of signals obtained after ajmaline infusion showing abnormal long-duration low frequency signals in the core of the electrophysiologically abnormal area (purple) and normal signals elsewhere. The proximal and distal signal is shown in each location.

More recently, this strategy, in combination with 3-dimensional duration maps, has been confirmed in a series of an additional 135 patients, proving to be more reliable in identifying patients at risk of VT/VF than risk stratification based on clinical presentation alone, including spontaneous ECG pattern, symptoms, or genetic or

family history.<sup>6</sup> Ajmaline infusion revealed a significant increase in BrS substrate size, even though most patients with the worst clinical presentation did not have a baseline spontaneous type 1 BrS ECG pattern, a family history of SCD at age < 45 years, or a positive test for *SCN5A*.<sup>6</sup> In our experience, the ability to accurately



**Figure 2.** Five consecutive ECGs showing progression of the ST-segment elevation, from basal, after ajmaline, during ablation, after ajmaline again and the final ECG. Note that precordial leads are presented as V<sub>1</sub>-V<sub>2</sub> in second (labeled V<sub>1</sub>-V<sub>2</sub>), V<sub>1</sub>-V<sub>2</sub> in third (labeled V<sub>3</sub>-V<sub>4</sub>) and V<sub>1</sub>-V<sub>2</sub> in the fourth intercostal spaces (labeled V<sub>5</sub>-V<sub>6</sub>) in each ECG. Note the increase in ST-elevation during radiofrequency (RF) delivery and how after ablation ST becomes rounded and is no longer coved-type.

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