Catheter Ablation for the Patient with Brugada Syndrome and Early Repolarization Syndrome

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The underlying electrophysiologic mechanism that causes an abnormal electrocardiogram (ECG) pattern and ventricular tachycardia/ventricular fibrillation (VT/VF) in patients with the Brugada syndrome (BrS) remains controversial. However, several studies have indicated that the right ventricular outflow tract (RVOT) is likely to be the site of electrophysiological substrate. My colleagues and I have found that in patients with BrS who have frequent recurrent VF episodes, the substrate site is indeed located at the RVOT but exclusively over the epicardium and not at the endocardium. Abnormal electrograms characterized by low-voltage fractionated late potential are present at the anterior RVOT epicardium of such BrS patient. More importantly, these abnormal electrograms are associated with epicardial surface and the interstitial fibrosis and reduced gap junction expression. Catheter ablation at these areas abolishes the BrS phenotype and life-threatening arrhythmias. BrS also associates with increased collagen throughout the heart. Thus it is very likely that abnormal myocardial structure and conduction are the underlying causes of electrophysiologic derangement in the BrS patients. The different findings were found in patients with early repolarization syndrome (ER) who had frequent VF episodes. In ER, trigger and initiator of VF are associated with abnormal Purkinje sites at the septal areas and inferior walls. There were no epicardial substrates unless there was a presence of concomitant BrS pattern. Catheter ablations of these triggers and abnormal Purkinje sites are effective in preventing VF recurrences. In my talks, I would discuss in details my experiences of catheter ablations in over 40 patients with BrS and 11 patients with ER as well as underlying pathophysiologic mechanisms of both BrS and ER.