

Purkinje-related Polymorphic Ventricular Tachycardia and Ventricular Fibrillation: Ablation of Just Trigger or Substrate Modification?

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There has been growing evidence that the Purkinje network plays a pivotal role in both the initiation and perpetuation of ventricular fibrillation (VF). A triggering ventricular premature beat (VPB) with a short-coupling interval could arise from either the right or left Purkinje system in patients with polymorphic ventricular tachycardia (VT) or VF, and that can be suppressed by the catheter ablation of the trigger. However, whether these ablations are just the suppression of trigger VPB or substrate modification is still unknown.

A focal breakdown in the “gating mechanism” at the Purkinje system resulting in a short-circuiting of the transmission across the gate at the distal Purkinje network might predispose to reentrant circuits of polymorphic VT/VF. Many investigators also reported the successful ablation of Purkinje-related VF with an acute or remote myocardial

infarction. The same approach with good short-term results has been reported in a small number of patients with other heart diseases (i.e. amyloidosis, chronic myocarditis, nonischemic cardiomyopathy, catecholaminergic polymorphic VT, Lamin A/C cardiomyopathy, left ventricular noncompaction). Catheter ablation of the triggering VPBs from the Purkinje system can be used as an electrical bailout therapy in patients with VF storm.