[Eng] Arrhythmia 3

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Beyond pulmonary vein isolation, the two main additional strategies: Cox-Maze procedure or targeting of electrical signatures (focal bursts, rotational activities, meandering wavelets), remain controversial. High-density mapping of these arrhythmias has demonstrated firstly that a patchy lesion set is highly proarrhythmogenic, favoring macro-re-entry through conduction slowing and providing pivots for localized re-entry. Secondly, discrete anatomical structures such as the Vein or Ligament of Marshall (VOM/LOM) and the coronary sinus (CS) have epicardial muscular bundles that are more frequently involved in re-entry than previously thought. The Marshall Bundle can be ablated at any point along its course from the mid-to-distal coronary sinus to the left atrial appendage. If necessary, the VOM may be directly ablated using ethanol infusion to eliminate PV contributions and produce conduction block across the mistral isthmus. Ethanol ablation of the VOM, supplemented with RF ablation, may be more effective in producing conduction block at the mitral isthmus than repeat RF ablation alone

[Eng] Arrhythmia 5

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Idiopathic ventricular fibrillation is diagnosed in patients who survived a ventricular fibrillation episode without any identifiable structural or electrical cause after extensive investigations. It is a common cause of sudden death in young adults. The study reviews the diagnostic value of systematic investigations and the new insights provided by detailed electrophysiological mapping. Recent studies have shown the high incidence of microstructural cardiomyopathic areas, which act as the substrate of ventricular fibrillation re-entries. These subclinical alterations require high-density endo- and epicardial mapping to be identified using electrogram criteria. Small areas are involved and located individually in various sites (mostly epicardial). Their characteristics suggest a variety of genetic or acquired pathological processes affecting cellular connectivity or tissue structure, such as cardiomyopathies, myocarditis, or fatty infiltration. Purkinje abnormalities manifesting as triggering ectopy or providing a substrate for re-entry represent a second important cause. The documentation of ephemeral Purkinje ectopy requires continuous electrocardiography monitoring for diagnosis. A variety of diseases affecting Purkinje cell function or conduction are potentially at play in their pathogenesis. Comprehensive investigations can therefore allow the great majority of idiopathic ventricular fibrillation to ultimately receive diagnoses of a cardiac disease, likely underlain by a mosaic of pathologies. Precise phenotypic characterization has significant implications for interpretation of genetic variants, the risk assessment, and individual therapy.