Identifying Sudden Cardiac Death Risk and Modeling a New Mechanism of Cardiac Conduction Responsible for Life Threatening Arrhythmias

Abstract: Since the advent of electrotherapies (pacemakers, implantable defibrillators, and cardiac ablation) for the treatment of cardiac arrhythmias, in lieu of pharmacotherapies, there has been a different focus on the identification of risk and approach to our mechanistic understanding of life threatening arrhythmias. Among the non-invasive risk stratifiers was the use of high resolution electrocardiography to record and quantify cardiac late potentials. In the experimental laboratory multi-electrode cardiac mapping provided insights into the sources of cardiac late potentials which eventually lead to the modern tools used in the clinical electrophysiology laboratory of today. Byproducts of these mapping approaches have been the details of direct cardiac recordings which have confounded the community with ambiguities which seemed to lack a biophysical basis, such as found in “fractionated” electrograms. As our understanding of cardiac cellular coupling grew in the area gap junction physiology, it was recognized that myo-fibroblasts were capable of forming gap junction connections with myocardial cells. These fibroblasts were often characterized as inert, structural cells, which responded to injury, e.g., myocardial infarction, to form scar tissue. However, their direct coupling provided us the opportunity to postulate a new mechanism of cardiac conduction. The “bridging” of myocardial cells via non-excitatable cells may facilitate conduction albeit with significantly slower conduction velocity. Tissue culture preparations and mathematical modeling were used to support the hypothesis of passive cell facilitation. These results have aided our understanding of new arrhythmia mechanisms but await final clinical confirmation.