## Biology and Pathology

## Cardiovascular Disease

### Introduction to Biology and Treatments: Electrophysiology and Atrial Fibrillation

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Learning Objectives:

- To understand multiple wavelets and focal activation theories
- To understand electrical, contractile and mechanical remodeling
- To understand intracellular Ca2+ handling abnormalities
- To understand modulating factors

Atrial fibrillation (AF) remains the most common adult rhythm disorder, and it associated with a substantial rate of morbidity and economic burden. The incidence of AF is expected to continue to rise with the aging of the population. AF involves a wide spectrum of arrhythmias from lone AF to paroxysmal to chronic AF. It is likely that AF comprises a spectrum of disease with no single mechanism adequate enough to comprehensively explain AF and its variability. Mechanism of fibrillation is explained by multiple wavelets and focal activation theories. Electrical, contractile and mechanical remodeling is involved in AF progression. Atrial remodeling may also increase in atrial fibrosis which can slow conduction velocity and can shorten the refractory period in atria with long-standing AF. Mechanical remodeling manifests as decreased atrial contractility and increased atrial compliance which leads to a stretch of the atrial myocardium. The importance of intracellular Ca2+ handling abnormalities has been highlighted, both for the induction of triggered ectopic activity and for the activation of Ca2+-related cell signaling that mediates profibrillatory remodeling. Modulating factors such as genetic factors, age, obesity, sleep apnea, inflammation, autonomic factors and atrial and pulmonary vein stretch only partially account for the increase in AF. Although significant progress in understanding the mechanism of this arrhythmia has been accomplished, the pathophysiology of AF is complex and likely has many possible mechanisms which may be interrelated.

***Key words: Atrial fibrillation; arrhythmia mechanism; remodeling; risk factor.***