

# Center for Cardiovascular Bioinformatics & Modeling

**Date: Wednesday, January 25, 2006**

**Time: 3:00 p.m.**

**Location: Clark Hall 110**

**Simulcast to Talbot Library**

## *Mechanisms of Atrial and Ventricular Fibrillation*

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### **Abstract:**

In the healthy heart, the wave of contraction that pumps the blood is created by a wave of electrical depolarization that passes from cell to cell through the myocardium. But in fibrillation, the depolarization wave breaks up into “electrical turbulence,” and coherent contraction is lost.

What causes this breakup? Cardiologists had long assumed that the cause of wavebreak must be external ‘wavebreakers,’ that is, areas of reduced conduction due to infarction, ischemia, fibrosis, etc. In contrast, physicists have shown that it is possible for a wave to break up due solely to the internal dynamics of wave propagation. Since the dynamics of wavefront formation and propagation depend in turn on ion channel dynamics within the cardiac myocytes, it follows that modifications of these ion channel dynamics could potentially stabilize the wave of propagation and prevent the breakup into fibrillation.

Our group at UCLA and Cedars-Sinai has pursued this strategy, using mathematical and physical theory together with very large scale models of cardiac propagation in realistic models of the atria and ventricles (a “Virtual Heart”). We have found that it is indeed possible to prevent the breakup into fibrillation by using drugs to modify ion-channel kinetics in the myocyte.

This talk will emphasize the mathematical and physical theory of wave conduction and wavebreak. We will present several equations, for wave propagation and for intracellular calcium dynamics, that play critical roles in the breakup that is fibrillation.

Our initial work on wave stability theory has since been supported by experiments in animal hearts, which confirm that modifying cell dynamics can serve as a potent anti-fibrillatory strategy, giving rise to new types of pharmacological and genetic interventions against atrial and ventricular fibrillation.