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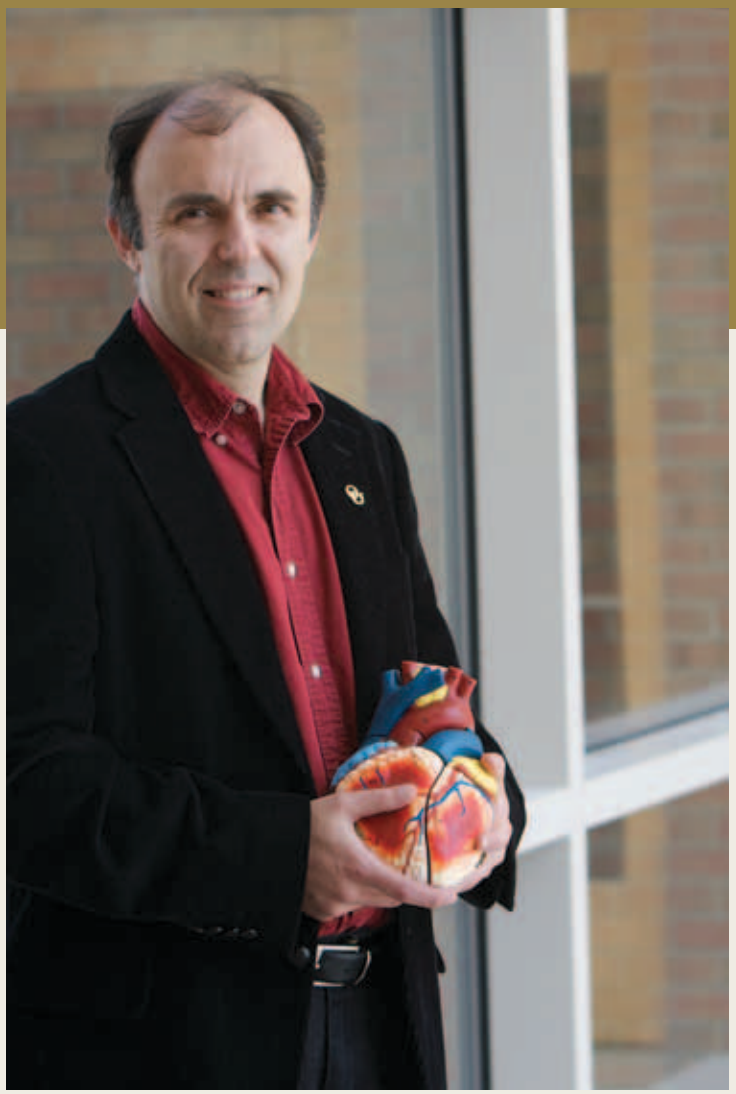
The shocking story of defibrillation



Brad Roth studies bioelectric phenomena, such as the electrical activity of the heart. The heart is an electrically controlled pump, and is normally controlled by regular organized waves of electrical activity. A severe and potentially fatal abnormality is ventricular fibrillation, where these waves propagate in a chaotic manner, like turbulence, preventing the heart from pumping blood. The way to treat fibrillation is to apply a strong electric shock: defibrillation. However, the detailed mechanism of how this shock defibrillates the heart is unknown. Roth's goal is to understand the fundamental physics that underlies defibrillation of the heart by analyzing how electric fields interact with cardiac tissue.

Roth uses mathematical modeling to simulate how the heart behaves. He performs sophisticated computer simulations to make predictions that can then be tested in the laboratory. Many of his calculations are based on the bidomain model, which is a mathematical model of the anisotropic electrical properties of cardiac tissue, consisting of a system of nonlinear partial differential equations. These numerical simulations indicate where the heart is stimulated, when it is stimulated, and how the resulting waves propagate and interact. This information is essential for understanding defibrillation.

One prediction of the bidomain model is the transmembrane potential distribution around an extracellular electrode injecting current, such as in a pacemaker. The transmembrane potential becomes positive (is depolarized) directly under the electrode. However, there also exist regions of negative transmembrane potential (hyperpolarization) adjacent to the electrode, along the fiber direction. During a strong shock, the regions of hyperpolarization affect both the mechanisms of wave excitation and the induction of a cardiac arrhythmia. For instance, if the tissue is depolarized just before the shock, the regions hyperpolarized by the shock may cause the tissue there to recover excitability, thereby creating an excitable path that can support wave propagation in one direction. A strong and well-timed shock can result in a type of arrhythmia called quatrefoil reentry, which is a simple model for ventricular fibrillation. Roth's calculations predicted the existence of quatrefoil reentry, which was subsequently observed in experiments using optical methods to measure the transmembrane potential.

Representative Recent Publications

1. Mazeh N, Roth BJ. 2009. A Mechanism for the upper limit of vulnerability. *Heart Rhythm* 6:361-367.

2. Prior P, Roth BJ. 2008. Calculation of optical signal using three-dimensional bidomain/diffusion model reveals distortion of the transmembrane potential. *Biophys* J 95:2097-2102.

3. Puwal S, Roth BJ. 2007. Forward Euler stability of the bidomain model of cardiac tissue. *IEEE Trans Biomed Eng* 54:951-953.

4. Janks DL, Roth BJ. 2006. Quatrefoil reentry caused by burst pacing. *J Cardiovasc Electrophysiol* 17:1362-1368.

5. Langrill Beaudoin D, Roth BJ. 2006. The effect of the fiber curvature gradient on break excitation in cardiac tissue. *PACE* 29:496-501.

6. Roth BJ. 2006. Defibrillators. In: *The Encyclopedia of Medical Devices and Instrumentation*, 2nd Edition. Webster JG, Ed, Wiley, Hoboken, NJ 2:406-410.