Chapter 2.1

The Bidomain Theory of Pacing

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Introduction

The implantable cardiac pacemaker is one of the most important medical innovations of the twentieth century. Yet until recently researchers have not understood the basic mechanisms governing how a pacemaker excites the heart. The development of a mathematical model describing the electrical properties of cardiac tissue – the bidomain model – helped unravel these mechanisms. This chapter outlines several important predictions of the bidomain model related to pacing. Several other chapters in this book examine related topics.

The bidomain model represents cardiac tissue as a multidimensional cable that can be represented by a network of resistors and capacitors. Figure 1 shows a network equivalent to the two-dimensional bidomain model. The lower grid of resistors represents the intracellular space, and the upper grid represents the extracellular space. The two spaces are coupled by resistors and capacitors representing the membrane. The electrical properties of cardiac muscle are markedly anisotropic; in Fig. 1 the resistors in the \( x \) direction may be different from the resistors in the \( y \) direction. Moreover, the degree of anisotropy differs within the intracellular and extracellular spaces. The ratio of conductivities in the \( x \) and \( y \) directions in the extracellular space is on the order of two, but in the intracellular space it is about ten, indicating the intracellular space is more anisotropic than the extracellular space. This condition of “unequal anisotropy ratios” leads to many of the interesting phenomena predicted by the bidomain model.

Unipolar Stimulation

Sepulveda et al. calculated the transmembrane potential in a passive two-dimensional sheet of cardiac tissue having unequal anisotropy ratios when a constant current is delivered

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I. R. Efimov et al. (eds.), Cardiac Bioelectric Therapy: Mechanisms and Practical Implications. © Springer Science+Business Media, LLC 2009
Make and Break Excitation

What is the mechanism by which an electrical current through a unipolar electrode excites cardiac tissue? Why can excitation occur near an anode as well as a cathode? Why can excitation be initiated by turning a stimulus off (break) as well as by turning it on (make)? The key to answering these questions is to realize that there are four distinct mechanisms responsible for the excitation of cardiac tissue: cathode make, anode make, cathode break, and anode break. These types of excitation have been known for decades, but the mechanism responsible for this behavior was not understood until the incorporation of an active membrane into the bidomain model.