INTERNATIONAL JOURNAL OF
NUMERICAL ANALYSIS AND MODELING
Volume 9, Number 3, Pages 644–657

AN ANALYSIS OF THE SHOCK STRENGTH NEEDED TO ACHIEVE DEFIBRILLATION IN A SIMPLIFIED MATHEMATICAL MODEL OF CARDIAC TISSUE

ASLAK TVEITO[1,2], GLENN LINES[1,2], MARIE E. ROGNES[1], AND MARY M. MALECKAR[1]

Abstract. The pumping function of the heart is driven by an electrical wave traversing the cardiac muscle in a well-organized manner. Perturbations to this wave are referred to as arrhythmias. Such arrhythmias can, under unfortunate circumstances, turn into fibrillation, which is often lethal. The only known therapy for fibrillation is a strong electrical shock. This process, referred to as defibrillation, is routinely used in clinical practice. Despite the importance of this procedure and the fact that it is used frequently, the reasons for defibrillation's effectiveness are not fully understood. For instance, theoretical estimates of the shock strength needed to defibrillate are much higher than what is actually used in practice. Several authors have pointed out that, in theoretical models, the strength of the shock can be decreased if the cardiac tissue is modeled as a heterogeneous substrate. In this paper, we address this issue using the bidomain model and the Courtemanche ionic model; we also consider a linear approximation of the Courtemanche model here. We present analytical considerations showing that for the linear model, the necessary shock strength needed to achieve defibrillation (defined in terms of a sufficiently strong change of the resting state) decreases as a function of an increasing perturbation of the intracellular conductivities. Qualitatively, these theoretical results compare well with computations based on the Courtemanche model. The analysis is based on an energy estimate of the difference between the linear solution of the bidomain system and the equilibrium solution. The estimate states that the difference between the linear solution and the equilibrium solution is bounded in terms of the shock strength. Since defibrillation can be defined in terms of a certain deviation from equilibrium, we use the energy estimate to derive a necessary condition for the shock strength.

Key Words. Defibrillation, Bidomain model, Energy estimate, Myocardial heterogeneities

1. Introduction

The beating of the human heart is a well-organized operation governed by an electrical wave which traverses the entire cardiac muscle to initiate contraction. This is a robust process, as it continues for about 80 years on average without maintenance, as well as versatile in the sense that it adapts smoothly to strongly varying external conditions. However, the heartbeat is not infallible. The regular electrical signal controlling the synchronous contraction of the heart may be disturbed. Such rhythm disturbances are known as arrhythmias and may result

Received by the editors January 25, 2011 and, in revised form, August 29, 2011.

in failure to adequately pump blood to the body. Ventricular fibrillation, an arrhythmia wherein the contraction of the heart muscle is completely asynchronous, is especially dangerous. If not treated within minutes, its ultimate consequence is sudden cardiac death [1].

Currently, the only effective means for prevention of sudden cardiac death is defibrillation of the heart by the timely application of a strong electric shock [2, 3, 4]. Significant advances have been made towards an improved understanding of the basic mechanisms by which a shock defibrillates the heart [5, 6, 7]. However, several key aspects of the interaction between an electric shock and the heart remain unclear. Hence, the mechanisms by which shocks terminate arrhythmias are far from fully understood. In recent years, mathematical modeling and simulation have had increasing importance in efforts to further understand the biophysical processes that underlie the generation of lethal arrhythmias and their termination via defibrillation [8, 9]. Simulations have offered not only increased understanding as to the mechanisms underlying defibrillation, but also valuable estimates of the amount of energy necessary to terminate fibrillation in a specific context [10, 11, 12].

Cardiac tissue, a functional syncytium, can be modeled mathematically via a continuum approximation, and fundamental ionic kinetics at the cell level by corresponding membrane models. However, when cardiac tissue is modeled via the bidomain approximation as a *homogeneous* substrate having uniform conductivities controlling current flow, simulations may result in an overestimation of the shock strength required for defibrillation [11]. Indeed, the assumption of homogeneity represents a simplification, as heterogeneities are realistic in terms of actual tissue structure. Such a simplification may yield results which are of questionable physiological relevance and limited clinical utility.

Microstructural myocardial heterogeneities have been previously represented in models through both random and localized alterations of conductivities and/or membrane kinetics [13, 14], as well as through realistic tissue discontinuities determined via high-resolution imaging [15, 16, 17]. The presence of larger-scale myocardial heterogeneities within computational bidomain models provides a substrate for bulk activation of the tissue following electric shocks, which may lower apparent estimates for defibrillation threshold. These larger-scale structures may include, for example, laminar clefts or vasculature, see [15, 18]. The inclusion of heterogeneities at multiple scales thus provides more realistic and lower estimates of the energy necessary to successfully defibrillate a particular substrate [19].

In this paper, we analyze the problem initially presented by Plank et al [20, 19]. These papers examine results obtained from simulations employing the bidomain model with cell membrane kinetics represented by the models of Courtemanche et al [21]. As outlined above, of particular interest is the result that the shock strength necessary to defibrillate decreases as tissue variability increases. Our aim is to contribute to a better understanding of this effect by providing theoretical estimates in addition to further numerical experiments.

The Courtemanche ionic model presents significant challenges to mathematical analysis. We have therefore introduced a linear approximation to the full model which we employ in its stead. In [22], it was demonstrated that this linear model provides fairly accurate solutions in the presence of strong shocks. In this paper, we present computational results examining the influence of random perturbations to the intracellular conductivities using both the full and the linear Courtemanche models. Furthermore, we provide analytical estimates for the linear model, showing that an increase in the variability of the intracellular conductivities leads to a