Published in final edited form as:
Biomech Model Mechanobiol. 2012 September ; 11(7): 995–1000. doi:10.1007/s10237-011-0368-1.

A Perturbation Solution Of The Mechanical Bidomain Model

Vanessa M. Punal and **Bradley J. Roth**
Department of Physics, Oakland University, Rochester, MI 48309

Vanessa M. Punal: vmpunal@oakland.edu; Bradley J. Roth: roth@oakland.edu

Abstract

This research focuses on finding analytical solutions to the mechanical bidomain model of cardiac tissue. In particular, a perturbation expansion is used to analyze the equations, with the perturbation parameter being inversely proportional to the spring constant coupling the intracellular and extracellular spaces. The results indicate that the intracellular and extracellular pressures are not equal, and that the two spaces can move relative to each other. This calculation is complicated enough to illustrate the implications of the mechanical bidomain model, but is nevertheless simple enough to solve analytically. One application of the calculation is to the mechanical behavior of active cardiac tissue surrounding an ischemic region.

Keywords

mechanical bidomain; cardiac tissue; perturbation expansion; pressure; stream function

Introduction

The mechanical behavior of cardiac tissue has been studied for decades. Nevertheless, the field remains vibrant because of new concepts such as mechanotransduction, and new experimental applications such as tissue engineering. Many of these issues concern the mechanical coupling of the intracellular and extracellular spaces. For instance, interactions between cells and the surrounding extracellular matrix occur in part through integrin proteins, which are thought to influence aspects of cellular function (Jean et al., 2011). In cardiac tissue, myocytes interact with the extracellular matrix mechanically, thereby regulating the remodeling of the tissue and cell structure (Kresh et al., 2011) – this is a type of mechanotransduction (McCain and Parker 2011). Engineered cardiac tissue is becoming increasingly important in clinical applications (Naito et al., 2006) and has been proposed as a tool to study cardiac ischemia (Katare et al., 2010). In particular, abnormal behavior at the border zone between ischemic and non-ischemic tissue can modulate tissue stress and strain, which could trigger remodeling (Rodriguez et al., 2005). The common theme among these various studies is a focus on the mechanical interaction between the intracellular and extracellular spaces. In order to understand these phenomena better, new theoretical models that highlight this interaction are required.

Puwal and Roth (2010), derived a mechanical bidomain model for cardiac tissue, analogous to the widely used electrical bidomain model (Henriquez, 1993). The bidomain model represents the tissue's intracellular and extracellular spaces individually as macroscopic continua and accounts for the coupling between the spaces with a spring constant. This model is a generalization of Ohayon and Chadwick's (1988) monodomain mechanical model, which contained three contributions to the stress: hydrostatic pressure, active fiber tension, and an extracellular collagen matrix. This model is a first step toward gaining a theoretical understanding of intracellular and extracellular mechanical interaction, but many implications of the model remain unexplained.

Puwal and Roth's (2010) analysis looked at simple examples, with displacements in one dimension. They found that the bidomain model makes two qualitative predictions: 1) the intracellular and extracellular pressures are not equal, and 2) it provides a new length constant which is small as K gets large. Our goal, in comparison, is to look at a more complicated two dimensional example that better illustrates the implications of the model but nevertheless is simple enough to solve analytically. In particular, we will focus on the problem of a region of ischemic tissue surrounded by healthy tissue (Latimer et al., 2003). This calculation is significant because alterations in strain at the ischemic border zone are important in their own right, and also because it allows us to further develop the mechanical bidomain model to better appreciate the role of coupling between the intracellular and extracellular spaces.

Methods

The mechanical bidomain equations describe the intracellular and extracellular displacements, $\boldsymbol{u}$ and $\boldsymbol{w}$, individually. We assume each space is incompressible, implying that each displacement has zero divergence $(\frac{\partial u_x}{\partial x}+\frac{\partial u_y}{\partial y}=0,\ \frac{\partial w_x}{\partial x}+\frac{\partial w_y}{\partial y}=0)$. As is commonly done for divergenceless displacements, we write $\boldsymbol{u}$ and $\boldsymbol{w}$ in terms of the stream functions ϕ and η $(u_x=\frac{\partial \phi}{\partial y},\ u_y=-\frac{\partial \phi}{\partial x},\ w_x=\frac{\partial \eta}{\partial y},\ w_y=-\frac{\partial \eta}{\partial x})$. In terms of these stream functions, the bidomain equations are:

$$\frac{\partial T}{\partial x}-\frac{\partial p}{\partial x}+\gamma\frac{\partial^3\phi}{\partial x^2\partial y}=K(u_x-w_x), \tag{1}$$

$$-\frac{\partial p}{\partial y}=K(u_y-w_y), \tag{2}$$

$$-\frac{\partial q}{\partial x}+\mu\left(\frac{\partial^3\eta}{\partial x^2\partial y}+\frac{\partial^3\eta}{\partial y^3}\right)=-K(u_x-w_x), \tag{3}$$

$$-\frac{\partial q}{\partial y}-\mu\left(\frac{\partial^3\eta}{\partial x\partial y^2}+\frac{\partial^3\eta}{\partial x^3}\right)=-K(u_y-w_y), \tag{4}$$

where p and q are the intracellular and extracellular pressure, $T(x, y)$ is the active tension of the fibers, and γ is the intracellular Young's modulus along the fiber length. We assume the fibers are uniform and in the x-direction. μ is the shear modulus of the isotropic extracellular space. Finally, K is a spring constant coupling the intracellular and extracellular spaces, and is a new parameter introduced in the bidomain model. Eqs. 1 through 4 say physically that the sum of the forces in the tissue (i.e. hydrostatic, active tension, extracellular shear, and intra-extracellular coupling) is zero.

We can simplify these equations by eliminating the intracellular/extracellular coupling. We take the x-derivative of Eq. 1 and the y-derivative of Eq. 2 and add them to obtain

$$\nabla^2 p=\frac{\partial^2 T}{\partial x^2}+\gamma\frac{\partial^4\phi}{\partial x^3\partial y}. \tag{5}$$

Similarly, we take the *x*-derivative of Eq. 3 and the *y*-derivative of Eq. 4 and add them to obtain

$$\nabla^2 q=0. \tag{6}$$

Alternatively, we can eliminate the hydrostatic pressures. We take the *y*-derivative of Eq. 1 and subtract the *x*-derivative of Eq. 2 to obtain

$$\frac{\partial^2 T}{\partial x\partial y}+\gamma\frac{\partial^4\phi}{\partial x^2\partial y^2}=K\nabla^2(\phi-\eta). \tag{7}$$

And, we take the *y*-derivative of Eq. 3 and subtract the *x*-derivative of Eq. 4 to obtain

$$\mu\nabla^4\eta=-K\nabla^2(\phi-\eta). \tag{8}$$

Our next task is to eliminate η from Eqs. 5 through 8 in favor of a new function λ, which we define as λ = ϕ − η. Our motivation for introducing λ is that we want to focus on differences between the intracellular and extracellular displacements. Furthermore, we will recast Eqs. 5 through 8 in terms of the dimensionless parameters $\varepsilon=\frac{\mu}{Ka^{2'}}\zeta=\frac{\gamma}{\mu'}$, and the dimensionless variables $X=\frac{x}{a}$ and $Y=\frac{y}{a}$, where *a* is a characteristic length for the problem. We obtain

$$\frac{a^2}{\mu}\nabla^2 p-\frac{a^2}{\mu}\frac{\partial^2 T}{\partial X^2}-\zeta\frac{\partial^4\phi}{\partial X^3\partial Y}=0, \tag{9}$$

$$\nabla^2 q=0, \tag{10}$$

$$\varepsilon\frac{a^2}{\mu}\frac{\partial^2 T}{\partial X\partial Y}+\varepsilon\zeta\frac{\partial^4\phi}{\partial X^2\partial Y^2}=\nabla^2\lambda, \tag{11}$$

$$\varepsilon\nabla^4(\phi-\lambda)=-\nabla^2\lambda. \tag{12}$$

We can also recast equations 1 through 4 in a similar manner.

Eqs. 1 through 4 along with 9 through 12 can be considered as alternative formulations of the fundamental equations of the bidomain model, which we will further analyze using perturbation theory. As noted by Puwal and Roth (2010), the coupling between the intracellular and extracellular space is strong, so *K* is large and we expect the dimensionless parameter ε to be small (ε ≪ 1). Therefore, we seek a solution that is a perturbation expansion in terms of ε

$$\phi=\phi_o+\varepsilon\phi_1+\cdots+\varepsilon^n\phi_n+\cdots, \tag{13}$$

$$\lambda=\lambda_o+\varepsilon\lambda_1+\cdots+\varepsilon^n\lambda_n+\cdots, \tag{14}$$

$$p=p_o+\varepsilon p_1+\cdots+\varepsilon^n p_n+\cdots, \tag{15}$$

$$q=q_o+\varepsilon q_1+\cdots+\varepsilon^n q_n+\cdots. \tag{16}$$

Substituting these expansions into Eqs. 1 through 4 and collecting terms with like powers of ε, we obtain the relations

zeroth − order (ε^0):

$$\frac{\partial \lambda_o}{\partial X}=\frac{\partial \lambda_o}{\partial Y}=0, \tag{17}$$

first − order (ε^1):

$$\frac{a^2}{\mu}\frac{\partial T}{\partial X}-\frac{a^2}{\mu}\frac{\partial p_o}{\partial X}+\zeta\frac{\partial^3 \phi_o}{\partial X^2 \partial Y}=\frac{\partial \lambda_1}{\partial Y}, \tag{18}$$

$$-\frac{a^2}{\mu}\frac{\partial p_o}{\partial Y}=-\frac{\partial \lambda_1}{\partial X}, \tag{19}$$

$$-\frac{a^2}{\mu}\frac{\partial q_o}{\partial X}+\left(\frac{\partial^3(\phi_o-\lambda_o)}{\partial X^2 \partial Y}+\frac{\partial^3(\phi_o-\lambda_o)}{\partial Y^3}\right)=-\frac{\partial \lambda_1}{\partial Y}, \tag{20}$$

$$-\frac{a^2}{\mu}\frac{\partial q_o}{\partial Y}-\left(\frac{\partial^3(\phi_o-\lambda_o)}{\partial X \partial Y^2}+\frac{\partial^3(\phi_o-\lambda_o)}{\partial X^3}\right)=\frac{\partial \lambda_1}{\partial X}. \tag{21}$$

Also, doing the same with Eqs. 9 through 12, we obtain

zeroth − order (ε^0):

$$\nabla^2 \lambda_o=0, \tag{22}$$

$$\frac{a^2}{\mu}\nabla^2 p_o-\frac{a^2}{\mu}\frac{\partial^2 T}{\partial X^2}-\zeta\frac{\partial^4 \phi_o}{\partial X^3 \partial Y}=0, \tag{23}$$

$$\nabla^2 q_o=0, \tag{24}$$

first − order (ε^1):

$$\frac{a^2}{\mu}\frac{\partial^2 T}{\partial X \partial Y}+\zeta\frac{\partial^4 \phi_o}{\partial X^2 \partial Y^2}=\nabla^2 \lambda_1, \tag{25}$$

$$\nabla^4(\phi_o-\lambda_o)=-\nabla^2 \lambda_1. \tag{26}$$

Given these equations, a four step procedure can be used to solve for the stream functions:

1. Eq. 17 implies that λ_o is a constant, which we take as zero

$$\lambda_o=0. \tag{27}$$

2. We add Eqs. 25 and 26, to obtain

$$\frac{a^2}{\mu}\frac{\partial^2 T}{\partial X\partial Y}+\zeta\frac{\partial^4\phi_o}{\partial X^2\partial Y^2}+\nabla^4\phi_o=0, \tag{28}$$

which allows us to solve for ϕ_o.

3. Once ϕ_o is known, we can solve Eq. 26 for λ_1

$$\nabla^2\lambda_1=-\nabla^4\phi_o. \tag{29}$$

4. Although above we have not carried out the analysis in terms proportional to ε^2, if we do so we find that once λ_1 is known, we can determine ϕ_1 by solving

$$\nabla^4\phi_1+\zeta\frac{\partial^4\phi_1}{\partial X^2\partial Y^2}=\nabla^4\lambda_1. \tag{30}$$

Once the stream functions are known, we can determine the pressures p and q from Eqs. 18 through 21, or from Eqs. 23 and 24.

Results

We demonstrate how to solve these equations using an example that is simple enough to analyze analytically, yet complicated enough to be non-trivial. In the region $x^2 + y^2 < a^2$, the tension is assumed to be

$$T=T_o\frac{(x^2+y^2)}{a^2}\left[2-\frac{(x^2+y^2)}{a^2}\right]=T_o(X^2+Y^2)\,[2-(X^2+Y^2)]. \tag{31}$$

This corresponds to zero tension at the origin, with the tension rising smoothly to T_o at $r = a$ where $r=\sqrt{x^2+y^2}$ (Fig. 1). This function was chosen because it approximates the case of a localized region of ischemia surrounded by healthy issue (Latimer et al., 2003).

At the boundary $r = a$, we assume the tissue is held in place, so $\boldsymbol{u} = \boldsymbol{w} = 0$. This is equivalent to saying that both ϕ and η have zero derivatives at the boundary, or in other words $\phi = \eta = 0$ and $\frac{\partial\phi}{\partial r}=\frac{\partial\eta}{\partial r}=0$ at $r = a$.

Step 1 of our four step process says $\lambda_o = 0$. Step 2 requires solving Eq. 28, which can be done analytically to find

$$\phi_o=\frac{a^2T_o}{3(3\zeta+16)\mu}XY(X^2+Y^2-1)^2. \tag{32}$$

Step 3 then gives

$$\lambda_1 = -\frac{32a^2 T_o}{3(3\zeta+16)\mu} XY(X^2+Y^2-1). \quad (33)$$

In this example $\Delta^4\lambda_1 = 0$, so step 4 and Eq. 30 yield

$$\phi_1 = 0. \quad (34)$$

The equation $\lambda = \phi - \eta$ tells us that $\eta_o = \phi_o$ and $\eta_1 = -\lambda_1$. Using Eqs. 18 and 19, we find that

$$p_o = -\frac{4T_o}{3(3\zeta+16)}[(\zeta+10)X^4 - (3\zeta+20)X^2 + 12X^2Y^2 - 4Y^2 + 2Y^4]. \quad (35)$$

In the same fashion, using Eqs. 20 and 21, we have

$$q_o = -\frac{4T_o}{3(3\zeta+16)}(Y^2 - X^2). \quad (36)$$

Figure 2 shows contour plots of ϕ_o and λ_1. In polar coordinates (r and θ), both functions vary as $\sin(2\theta)$. In a contour plot of a stream function, the displacement is parallel to the isocontour lines. Figure 3a shows the displacement $\boldsymbol{u}$ to zeroth order (which is the same as $\boldsymbol{w}$) as a vector field plot. Figure 3b contains a vector plot of the difference between the intracellular and extracellular displacements, $\boldsymbol{u} - \boldsymbol{w}$, which is only non-zero in first order. Note that the vectors in Fig. 3b should actually have a much shorter length than those in Fig. 3a because of the factor of ε in the perturbation expansion (although the factor of 32 in Eq. 33 partially offsets the effect of the factor of ε). However, we have rescaled the vectors to have a similar size in both plots so that the difference field is easier to visualize. Figures 4a and 4b show the intracellular and extracellular pressures, respectively, as a function of position. Note that the two pressures have very different spatial distributions, and the intracellular pressure is positive everywhere and large, whereas the extracellular pressure varies between positive and negative, and is small.

Discussion

Many experimental studies are focusing on the biomechanical interactions of the intracellular and extracellular spaces, and the only multicellular theoretical model that accounts for the intracellular and extracellular spaces individually is the mechanical bidomain model (Puwal and Roth, 2010). In this study, we extend the bidomain model and explore its implications. Specifically, we have 1) rewritten the mechanical bidomain equations in terms of stream functions and two dimensionless parameters ζ and ε, 2) identified the parameter ε as small and introduced a perturbation expansion, 3) derived a four-step procedure for solving the lowest-order contributions to the mechanical bidomain equations, and 4) solved these equations analytically for the simple example of a localized region of ischemic tissue.

The first step of our four-step procedure implies that the lowest-order contribution to the stream function λ vanishes, $\lambda_o = 0$. Because λ is the difference between the intracellular and extracellular stream functions, this result implies that to zeroth-order the intracellular and extracellular spaces move together ($\boldsymbol{u} = \boldsymbol{w}$). Another way to interpret this is that a

monodomain model (which treats the tissue as a single material with the intracellular and extracellular spaces averaged together) is sufficient to describe the mechanical behavior of the tissue to zeroth-order. Moreover, Eq. 28 in the second step of our four-step procedure is the monodomain equation equivalent to that derived by Ohayon and Chadwick (1988), containing an active tension, a Young's modulus along the fibers, and an isotropic collagen matrix. Although Eq. 28 does not depend on ε, it does depend on ζ, which indicates the relative importance of the fiber Young's modulus and the collagen shear modulus. Ohayon and Chadwick (1988) estimated ζ to be approximately 3. Latimer et al. (2003) solved a monodomain problem of local ischemia using $\zeta = 0$. Our bidomain model therefore improves on the Latimer et al. (2003) result in two ways: it has nonzero ζ and it includes the bidomain behavior.

Our results in Figure 4 show that the intracellular and extracellular spaces have different pressures, both in magnitude and spatial distribution. This result, which was also noted by Puwal and Roth (2010), appears even in the zeroth-order; it is not a small effect. Presumably if this difference is maintained for an extended period of time the pressure difference would drive fluid from one space to another. Interestingly, the intracellular pressure is larger than the extracellular pressure, and at some locations the extracellular pressure can even be negative. Therefore, we would expect fluid to be driven from the intracellular space into the extracellular space.

The impact of the bidomain model on the displacement becomes evident in the third step of our four-step procedure. Eq. 29 governs λ_1, the lowest-order nonzero difference between the intracellular and extracellular stream functions. Figure 3b shows where in the tissue the intracellular space is displaced relative to the extracellular space. If ε <<1, then this displacement will be small. What is not clear from our model is how this relative motion will affect the stresses in the cell membrane. One would expect that where the two spaces move relative to each other is where the membrane would undergo the greatest stress. For example, stretch-induced ion channels (Kohl and Sachs, 2001) are thought to play an important role in the electrical behavior of cardiac tissue. Monodomain models often suggest that these channels respond to fiber strain, $\varepsilon_{xx}=\frac{\partial^2\phi_o}{\partial X\partial Y}$ (Latimer et al., 2003). The bidomain model implies a different hypothesis: the channels respond to the difference in displacements in the two spaces, $\boldsymbol{u} - \boldsymbol{w}$. These two functions have very different distributions. For instance, in our analytical example, at the center of the tissue ε_{XX} is large while $\boldsymbol{u} - \boldsymbol{w}$ vanishes. Therefore, the location of stretch-induced ion channel activation is very different in the monodomain and bidomain models. In fact, one could argue that any transmembrane mechanical signaling may respond to $\boldsymbol{u} - \boldsymbol{w}$, and therefore require a bidomain model for analysis.

In our example, we used a boundary condition of $\boldsymbol{u} = \boldsymbol{w} = 0$ at the tissue edge, $r = a$. Other boundary conditions exist, such as having one or both spaces free (zero normal stress). This issue of boundary conditions becomes important when using perturbation theory. In Eqs. 11 and 12, the highest derivatives are multiplied by ε, which is a characteristic of singular perturbation theory (Van Dyke, 1964; Johnson, 2005). In general, Laplace's equation requires only one boundary condition, but on physical grounds we expect two boundary conditions to apply in Eq. 29:$\lambda_1 = 0$ and $\frac{\partial\lambda_1}{\partial r}=0$. We solved Eq. 29 using the boundary condition $\lambda_1 = 0$, which implies that tangential displacements do not vanish at $r = a$ (see Fig. 3b). We suspect that solving the bidomain equations (Eqs. 11 and 12) subject to both boundary conditions would require the methods of singular perturbation theory, and would introduce a thin boundary layer near $r = a$ (Van Dyke, 1964). This is not unexpected, as

Puwal and Roth (2010) found that the mechanical bidomain model introduces a new length scale inversely proportional to $\sqrt{K}$, which in our notation is proportional to $\sqrt{\varepsilon}$.

With any new theoretical model, one key question is: what new experimental predictions does it make? The mechanical bidomain model suggests several interesting experiments. 1) Simultaneous measurement of intracellular and extracellular displacements could be compared with theoretical predictions. These measurements would need to be very accurate, as differences between the two displacements may be small. 2) The introduction of λ into the bidomain equations suggests an analogous experimental approach, in which experimental methods are developed that measure directly the difference between the intracellular and extracellular displacements. We are not certain how such a measurement would be done, but if some method were available to measure distances on a nanometer scale (perhaps by fluorescence resonance energy transfer, FRET) between molecules on the inside and outside of the membrane, then $\boldsymbol{u} - \boldsymbol{w}$ could be imaged directly. 3) The model predicts different hydrostatic pressures in the intracellular and extracellular spaces. If these two pressures could be measured independently, this model prediction could be tested. One problem would be separating out the hydrostatic pressure contributions to the stress from other contributions. If pressures could not be measured easily, then one could search for water flow across the membrane, driven by the pressure difference. 4) If stretch-induced ion channels could be monitored individually (perhaps by patch clamping), then one could test the hypothesis that the membrane responds to differences in the intracellular and extracellular displacement, rather than other quantities such as fiber strain. 5) One could image the tissue near a fixed boundary to attempt to detect the formation of a thin mechanical boundary layer. 6) By using drugs or other methods, one could vary K by decoupling the intracellular and extracellular spaces, and compare the resulting behavior to bidomain predictions. Such an experiment might be easiest using thin tissue layers grown on an extracellular substrate. All of these experiments would be difficult, but they all follow from theoretical predictions from the mechanical bidomain model.

There are limitations to our findings and predictions. We have assumed that fibers are straight and uniform. In our work we have taken γ = constant, while Ohayon and Chadwick (1988) allowed γ to vary with active tension. In our model the intracellular and extracellular spaces are individually incompressible ($\nabla \cdot \boldsymbol{u} = \nabla \cdot \boldsymbol{w} = 0$), whereas in general the two spaces together are incompressible but individually they may not be. We have assumed small displacements and used a linear approximation for the strain. Perhaps most importantly, the active tension and tissue geometry we assumed in our example are artificial and overly simplified. Finally, we do not have a good measurement of K, so we are not sure how small ε is. Despite these limitations, this calculation has the virtue of being analytically solvable, it makes several new predictions that can be tested experimentally, and it illustrates clearly how perturbation methods can be used to account for bidomain effects. The development of numerical methods that can solve more realistic problems, and the use of singular perturbation theory to account for the boundary conditions, are two problems that require additional study.

Acknowledgments

This research was supported by The National Institutes of Health (R01 EB 008421). We thank Steffan Puwal for his comments and suggestions.

References

Henriquez CS. Simulating the electrical behavior of cardiac tissue using the bidomain model. Crit. Rev. Biomed. Eng. 1993; 21(1):1–77. [PubMed: 8365198]

Jean C, Gravelle P, Fournie JJ, Laurent G. Influence of stress on extracellular matrix and integrin biology. Oncogene. 2011; 30(24):2697–2706. [PubMed: 21339741]

Johnson, RS. Singular perturbation theory – mathematical and analytical techniques with applications to engineering. NY: Springer; 2005.

Katare RG, Ando M, Kakinuma Y, Sato T. Engineered heart tissue: a novel tool to study ischemic changes of the heart in vitro. PLoS One. 2010; 5(2):e9275. [PubMed: 20174664]

Kresh JY, Chopra A. Intercellular and extracellular mechanotransduction in cardiac myocytes. Pflugers Archiv. European J. Phys. 2011; 462(1):75–87. [PubMed: 21437600]

Kohl P, Sachs F. Mechanoelectric feedback in cardiac cells. Philos. Trans. R. Soc. Lond. B Biol. Sci. 2001; 359:1173–1185.

Latimer DC, Roth BJ, Parker KK. Analytical model for predicting mechanotransduction effects in engineered cardiac tissue. Tissue Eng. 2003; 9:283–289. [PubMed: 12740090]

McCain ML, Parker KK. Mechanotransduction: the role of mechanical stress, myocytes shape, and cytoskeletal architecture of cardiac function. Pflugers Archiv. European J. Phys. 2011; 462(1):89–104. [PubMed: 21499986]

Naito H, Melnychenko I, Didie M, Schneiderbanger K, Schubert P, Rosenkranz S, Eschenhagen T, Zimmermann W. Optimizing engineered heart tissue for therapeutic applications as surrogate heart muscle. Circulation. 2006; 114:I-72–I-78. [PubMed: 16820649]

Ohayon J, Chadwick RS. Effects of collagen microstructure on the mechanics of the left ventricle. Biophys. J. 1988; 54:1077–1088. [PubMed: 3233266]

Puwal S, Roth BJ. Mechanical bidomain model of cardiac tissue. Phys. Rev. E. 2010; 82 041904.

Rodriguez F, Langer F, Harrington KB, Cheng A, Daughters GT, Criscione JC, Ingels NB, Miller DC. Cardiopulmonary support and physiology alterations in transmural strains adjacent to ischemic myocardium during acute midcircumflex occlusion. J. Thorac Cardiovasc Surg. 2005; 129:791–803. [PubMed: 15821645]

Van Dyke, M. Perturbation methods in fluid mechanics. NY: Academic Press; 1964.

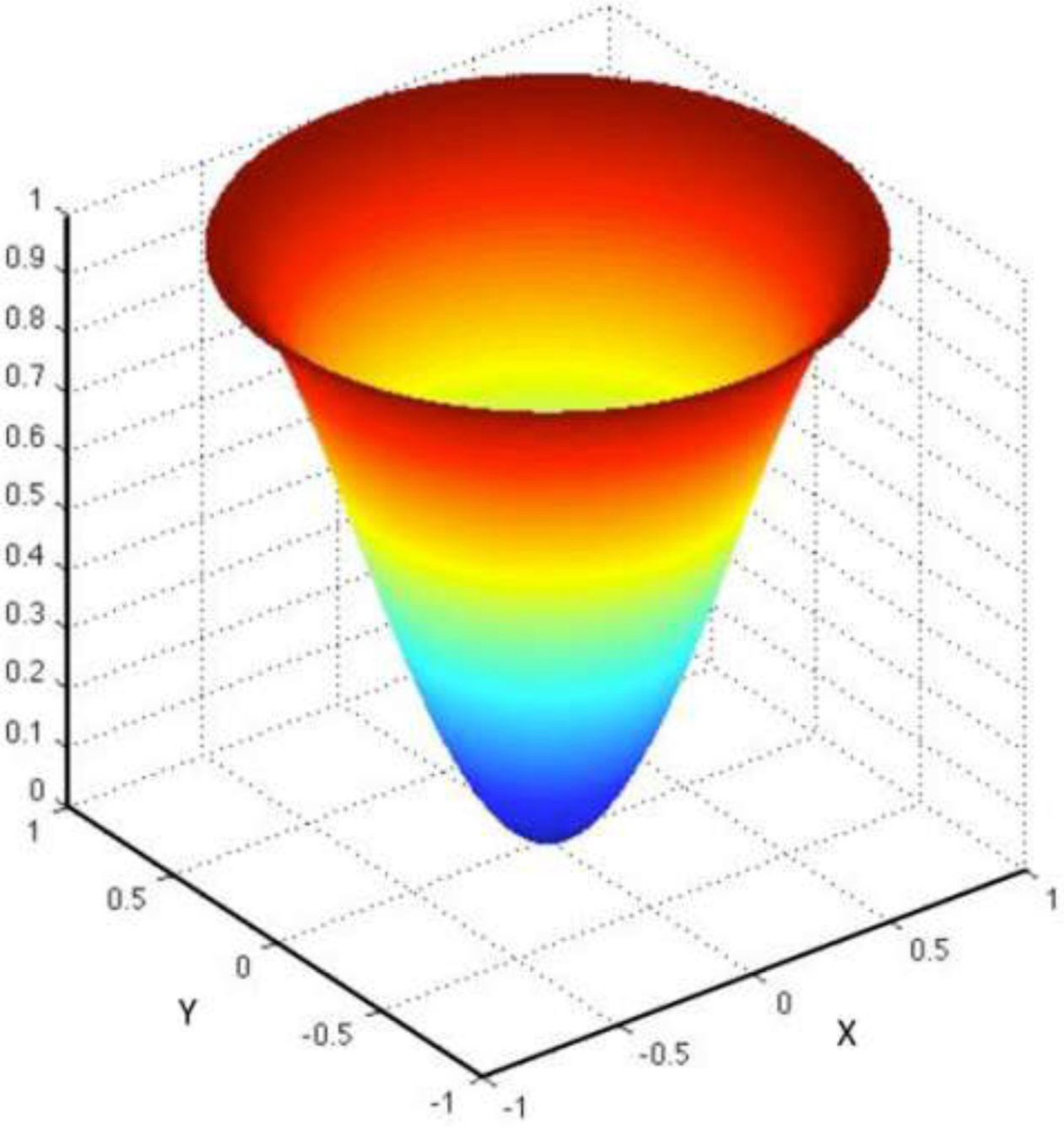
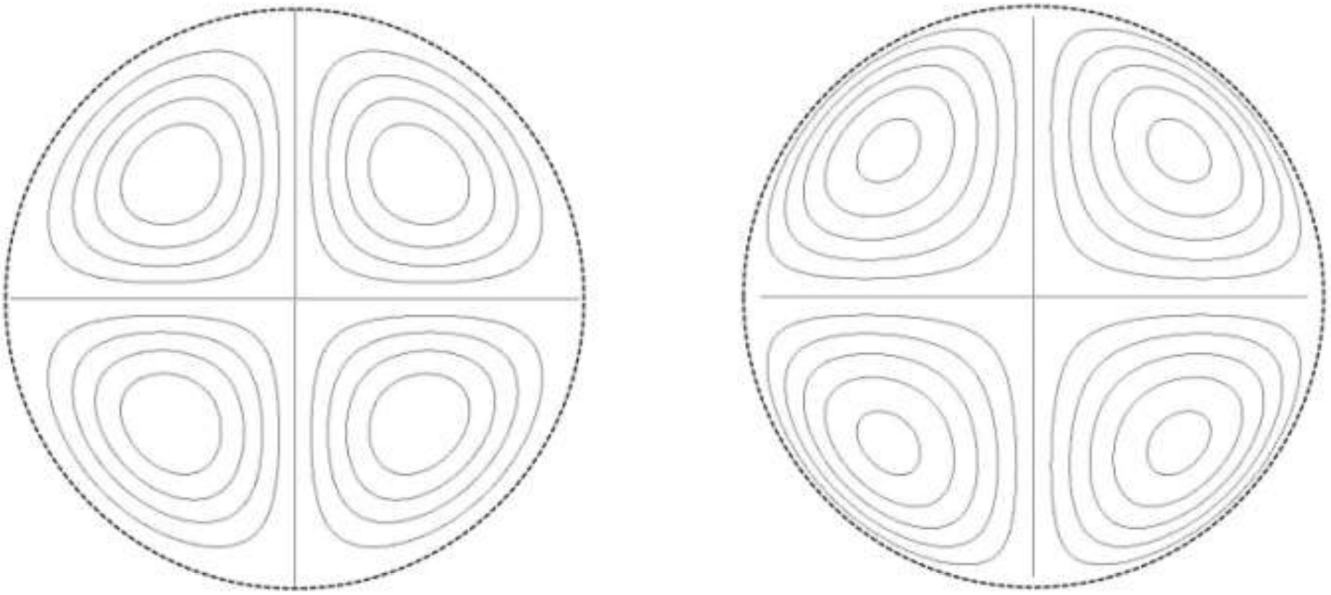


Fig 1.

The active tension $\frac{T}{T_o}$ as a function of X and Y, given by Eq. 31.



(a) (b)

Fig. 2.
Contour plots of a) the intracellular stream function to zeroth order (Eq. 32) and b) the difference of the intracellular and extracellular stream functions, to first order (Eq. 33). The fibers are horizontal.

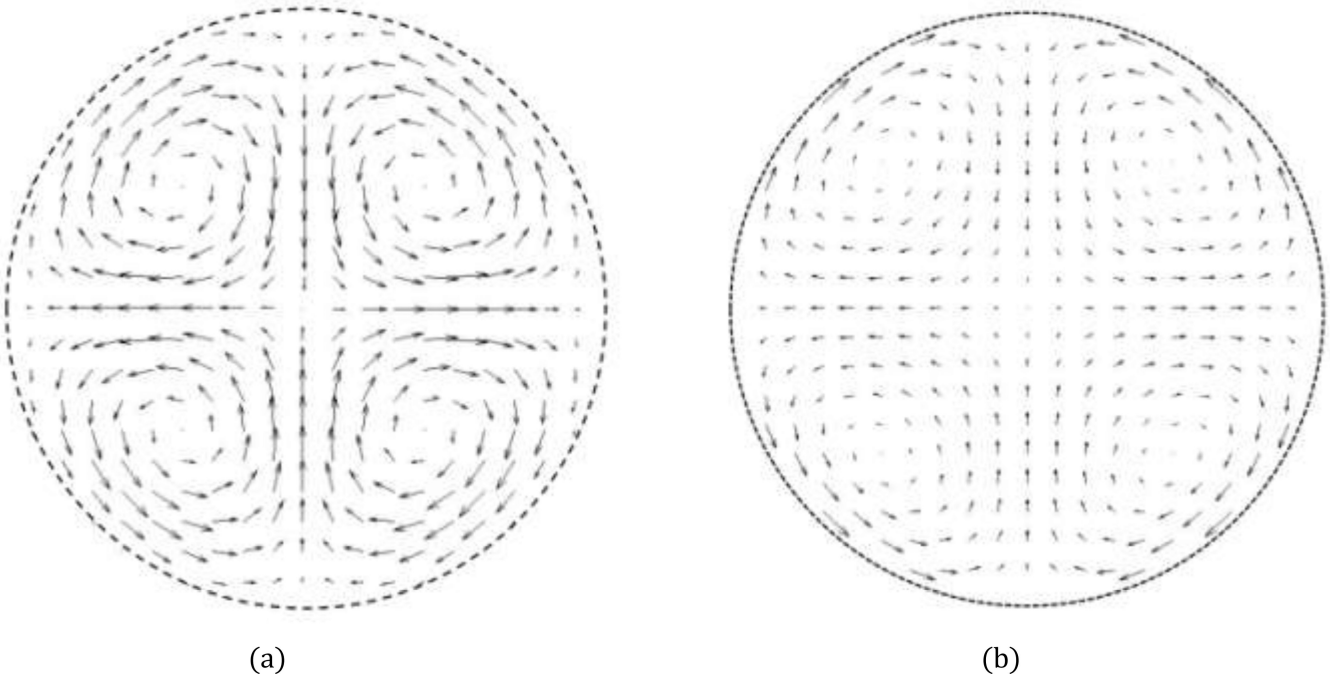
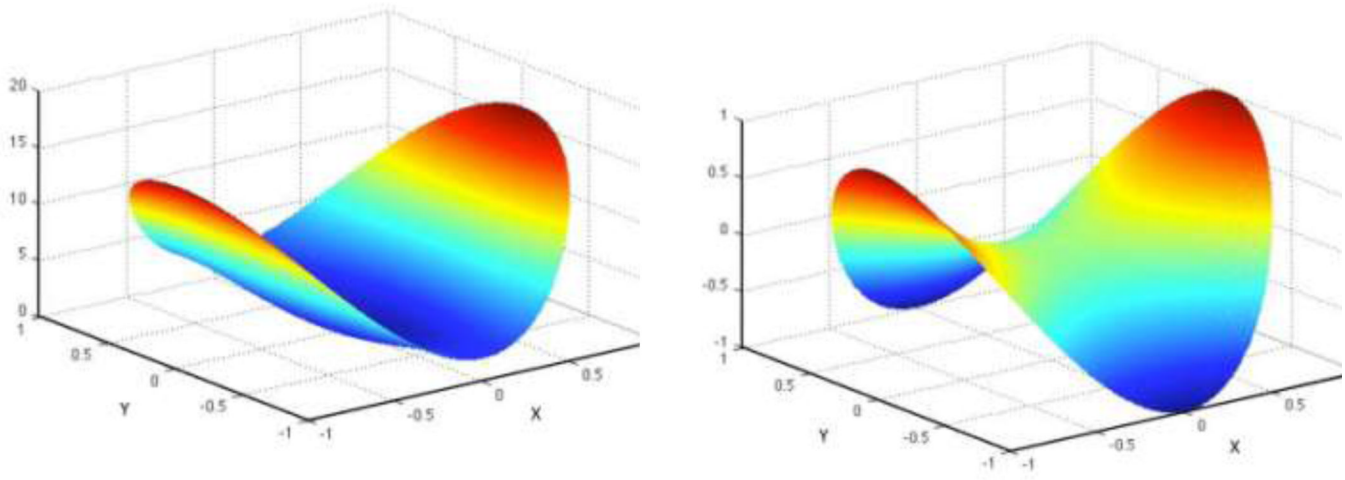


Fig. 3.

Vector plots of a) the intracellular displacement to zeroth order, and b) the difference of the intracellular and extracellular displacements to first order. The fibers are horizontal. The scaling of the vector magnitude is arbitrary, and different in a) and b).



(a) (b)

Fig. 4.

Plots of the a) intracellular and b) extracellular pressure as a function of X and Y (Eqs. 35 and 36). The amplitude of each pressure is divided by $\frac{4T_o}{3(3\zeta+16)}$. The fibers are parallel to the X axis, and $\zeta = 3$.