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Boundary Layers and the Distribution of Membrane Forces Predicted by the Mechanical Bidomain Model

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Abstract

The mechanical bidomain model is a mathematical description of the elastic properties of cardiac tissue. The unique feature of the bidomain model is that it is a macroscopic continuum representation of tissue that nevertheless accounts for the intracellular and extracellular spaces individually, thereby focusing on mechanical forces arising across the cell membrane. In this paper, the mechanical bidomain model describes a two-dimensional sheet of cardiac tissue undergoing a uniform active tension. At the boundary, the tissue sheet is free to move. Analytical solutions are found for the intracellular and extracellular displacements and pressures. The model predicts that membrane forces, which may be responsible for phenomena such as mechanotransduction and remodeling, are large near the tissue boundary, and fall off rapidly with distance from the boundary.

Keywords

mechanical bidomain model; displacement; boundary layer; cardiac tissue

1. Introduction

The mechanical bidomain model is a mathematical description of the elastic properties of cardiac tissue (Puwal and Roth, 2010; Punal and Roth, 2012; Roth, 2013). The mechanics of the heart has been studied for decades, and sophisticated models exist that predict the displacement, pressure, stress, and strain (Arts et al., 1979; Ohayon and Chadwick, 1988; Guccione et al., 1991; Glass et al., 1991; Rodriguez et al., 1994; Humphrey, 2002). The unique feature of the bidomain model is that, although it is a macroscopic model describing the behavior of the tissue averaged over many cells, it nevertheless accounts for the intracellular and extracellular spaces individually. This property lets one focus on mechanical forces arising across the membrane caused by different displacements in the two spaces. The fundamental hypothesis that underlies and motivates the mechanical bidomain model is that membrane forces are important for many biological phenomena, such as remodeling and mechanotransduction (Kresh and Chopra, 2011; McCain and Parker, 2011; Jean et al., 2011; Dabiri et al., 2012). Traditional “monodomain” models of cardiac mechanics do not predict where these membrane forces occur.

The mechanical bidomain model is similar in structure to biphasic models used in biomechanics (Mow et al., 1984; Ateshian et al., 1997; Humphrey, 2003). For instance, the equations of the mechanical bidomain model have a structure very similar to Eqs. 2 and 3 published by Ateshian et al. (1997) to describe cartilage. However, their model is a fluid-solid mixture, whereas the mechanical bidomain model represents a mixture of two elastic

solids. The coupling in their model arises from friction between the relative motion of the fluid and solid phases, whereas in my model the coupling arises from an elastic coupling between the two solid phases. I have adopted the terminology of the “intracellular space” and “extracellular space” because of its analogy to the terminology common in the electrical bidomain model (itself a mixture model). However, one could alternatively use terminology more common to the biomechanics literature, and refer to the mechanical bidomain model as “biphasic model,” a mixture of an “intracellular phase” and an “extracellular phase”.

When developing a new model, analytical solutions to simple and idealized problems are useful for developing insight and exploring new qualitative behavior. In this paper, I apply the bidomain model to an elementary yet fundamental problem: a two-dimensional circular sheet of cardiac tissue undergoing an active tension acting along the myocardial fibers, which are assumed to be uniform and straight. At the boundary, the tissue sheet is free (zero normal stress). In such a situation, the tissue should contract along the fibers, and—because it is incompressible—bulge outward perpendicular to the fibers. In this case, where are the membrane forces largest? The answer to this question is not obvious, and is in fact the central question that I address.

2. Methods

The intracellular stress consists of three terms: a hydrostatic pressure p that arises because the tissue is mostly water, a shear stress characterized by the intracellular shear modulus ν , and an active tension T along the fibers (Ohayon and Chadwick, 1988), which I assume are oriented parallel to the x -axis. The contribution of the active tension to the stress tensor would be easiest to express in Cartesian coordinates. However, I consider a circular sheet of radius R , and the application of the boundary conditions at $r = R$ is simplest in cylindrical coordinates. The intracellular stress tensor, σ_i , is therefore (Latimer et al., 2003)

$$\sigma_{irr} = -p + 2\nu\epsilon_{irr} + \frac{T}{2}(1 + \cos 2\theta) \quad (1)$$

$$\sigma_{i\theta\theta} = -p + 2\nu\epsilon_{i\theta\theta} + \frac{T}{2}(1 + \cos 2\theta) \quad (2)$$

$$\sigma_{ir\theta} = 2\nu\epsilon_{ir\theta} - \frac{T}{2}\sin 2\theta, \quad (3)$$

where ϵ_i is the intracellular strain tensor. For simplicity we do not include a Young's modulus term along the fibers as was used previously (Puwal and Roth, 2010; Punal and Roth, 2012), which means that the intracellular shear stress/strain relationship is isotropic¹. The extracellular stress tensor σ_e is expressed similarly, except that there is no active tension

$$\sigma_{err} = -q + 2\mu\epsilon_{err} \quad (4)$$

$$\sigma_{e\theta\theta} = -q + 2\mu\epsilon_{e\theta\theta} \quad (5)$$

$$\sigma_{er\theta} = 2\mu\epsilon_{er\theta}, \quad (6)$$

¹Previous studies with the mechanical bidomain model did not include an isotropic shear modulus in the intracellular space ($\nu=0$). However, ν plays a crucial role in this calculation. For example, the thickness of the boundary layer depends on ν (Eq. 21).

where ϵ_e is the extracellular strain tensor, q is the extracellular pressure, and μ is the extracellular shear modulus.

In cylindrical coordinates, the strain tensors are given in terms of the intracellular and extracellular displacements, $\mathbf{u}$ and $\mathbf{w}$, by (Love, 1944)

$$\epsilon_{irr} = \frac{\partial u_r}{\partial r} \quad \epsilon_{err} = \frac{\partial w_r}{\partial r} \quad (7)$$

$$\epsilon_{i\theta\theta} = \frac{u_r}{r} + \frac{1}{r} \frac{\partial u_\theta}{\partial \theta} \quad \epsilon_{e\theta\theta} = \frac{w_r}{r} + \frac{1}{r} \frac{\partial w_\theta}{\partial \theta} \quad (8)$$

$$\epsilon_{ir\theta} = \frac{1}{2} \left(\frac{1}{r} \frac{\partial u_r}{\partial \theta} + \frac{\partial u_\theta}{\partial r} - \frac{u_\theta}{r} \right) \quad \epsilon_{er\theta} = \frac{1}{2} \left(\frac{1}{r} \frac{\partial w_r}{\partial \theta} + \frac{\partial w_\theta}{\partial r} - \frac{w_\theta}{r} \right). \quad (9)$$

Here, I assume small displacements, so the linear approximation for the strain applies. Furthermore, I assume plane strain, so there are no displacements in the z direction perpendicular to the plane of the sheet. Finally, I assume both spaces are individually incompressible ($\nabla \cdot \mathbf{u} = \nabla \cdot \mathbf{w} = 0$). In that case, the displacements can be found from intracellular and extracellular stream functions ϕ and η (Love, 1944)

$$u_r = -\frac{1}{r} \frac{\partial \phi}{\partial \theta} \quad w_r = -\frac{1}{r} \frac{\partial \eta}{\partial \theta} \quad (10)$$

$$u_\theta = \frac{\partial \phi}{\partial r} \quad w_\theta = \frac{\partial \eta}{\partial r}. \quad (11)$$

The equations of static equilibrium consist of two equations for the intracellular space and two for the extracellular space. Each of these equations includes terms that arise from the divergence of the stress tensor (Love, 1944), and a term unique to the bidomain model that represents the coupling between the intracellular and extracellular spaces

$$\frac{\partial \sigma_{irr}}{\partial r} + \frac{1}{r} \frac{\partial \sigma_{ir\theta}}{\partial \theta} + \frac{\sigma_{irr} - \sigma_{i\theta\theta}}{r} = K(u_r - w_r) \quad (12)$$

$$\frac{\partial \sigma_{ir\theta}}{\partial r} + \frac{1}{r} \frac{\partial \sigma_{i\theta\theta}}{\partial \theta} + 2 \frac{\sigma_{ir\theta}}{r} = K(u_\theta - w_\theta) \quad (13)$$

$$\frac{\partial \sigma_{err}}{\partial r} + \frac{1}{r} \frac{\partial \sigma_{er\theta}}{\partial \theta} + \frac{\sigma_{err} - \sigma_{e\theta\theta}}{r} = -K(u_r - w_r) \quad (14)$$

$$\frac{\partial \sigma_{er\theta}}{\partial r} + \frac{1}{r} \frac{\partial \sigma_{e\theta\theta}}{\partial \theta} + 2 \frac{\sigma_{er\theta}}{r} = -K(u_\theta - w_\theta). \quad (15)$$

I assume the two spaces are coupled by a Hookean spring, with spring constant K , representing the force acting across the cell membrane (Puwal and Roth, 2010). This force most likely arises because of different displacements on the intracellular and extracellular ends of membrane spanning proteins, which cause the proteins to be distorted mechanically. The membrane force acts on the intracellular space with positive sign, and the extracellular

space with negative sign, forming an action/reaction pair. No external body forces are present.

Both spaces are individually stress free at the edge of the tissue sheet. More specifically, the boundary conditions at $r = R$ are that

$$\sigma_{irr} = \sigma_{ir\theta} = \sigma_{err} = \sigma_{er\theta} = 0. \quad (16)$$

The active tension T is generally a function of position (Punal and Roth, 2012), but in this analysis I take the simplest nontrivial form for T , a constant.

3. Results

The solutions for the stream functions and pressures are

$$\phi = AR^2 \sin 2\theta + CI_2\left(\frac{r}{\lambda}\right) \sin 2\theta \quad (17)$$

$$\eta = Br^2 \sin 2\theta - \frac{\nu}{\mu} CI_2\left(\frac{r}{\lambda}\right) \sin 2\theta \quad (18)$$

$$p = Dr^2 \cos 2\theta + \frac{T}{2} \quad (19)$$

$$q = -Dr^2 \cos 2\theta. \quad (20)$$

The expressions for the stream functions (Eqs. (17) and (18)) each contain two terms. The first is proportional to r^2 and represents the expected contraction parallel to the fibers and extension perpendicular to them. The second term has the form of a boundary layer, which is typical for solutions to the homogeneous mechanical bidomain equations (Roth and Puwal, 2010; Roth, 2012; Roth, 2013). I_2 is a modified Bessel function, which rises monotonically with r , similar to an exponential. It is therefore large at the tissue edge and falls off rapidly with depth into the tissue, with a length constant λ .

$$\lambda = \sqrt{\frac{\mu\nu}{K(\mu+\nu)}}. \quad (21)$$

The constants A , B , C , and D can be found from the boundary conditions

$$A = \frac{T}{8(\mu+\nu)} \left[1 + 4\left(\frac{\lambda}{R}\right)^2 \frac{\nu}{\mu} f(R) \right] \quad (22)$$

$$B = \frac{T}{8(\mu+\nu)} \left[1 - 4\left(\frac{\lambda}{R}\right)^2 \left(\frac{\nu}{\mu}\right)^2 f(R) \right] \quad (23)$$

$$C = \frac{T}{8(\mu+\nu)} \left(\frac{\mu}{\nu}\right) \frac{1}{h(R)} \left[1 - 4\left(\frac{\lambda}{R}\right)^2 \left(\frac{\nu}{\mu}\right)^2 f(R) \right] \quad (24)$$

$$D = \frac{T}{2} \frac{\nu}{\mu + \nu} \left(\frac{\nu}{\mu} \right) \frac{1}{R^2} f(R), \quad (25)$$

where $f(R)$ is

$$f(R) = \frac{1 - \frac{g}{h}}{1 + 4 \left(\frac{\lambda}{R} \right)^2 \left(1 - \frac{g}{h} \right)} \quad (26)$$

and

$$g(R) = -\frac{1}{r^2} I_2 + \frac{1}{r} \frac{dI_2}{dr} \quad (27)$$

$$h(R) = \frac{2}{r^2} I_2 + \frac{1}{4\lambda^2} I_2 - \frac{1}{2r} \frac{dI_2}{dr}, \quad (28)$$

with I_2 and dI_2/dr evaluated at $r = R$.

To make sense of these rather complicated expressions, consider the limit of a thin boundary layer. The value for K is not known, so the size of λ is uncertain. However, K should be large (the intracellular and extracellular spaces are tightly coupled, so they cannot slide past each other easily), implying that λ is small, $\lambda \ll R$. In that case, f is approximately equal to

one and h is approximately equal to $\frac{1}{4\lambda^2} I_2 \left(\frac{R}{\lambda} \right)$, so in this approximation

$$\phi = \frac{T}{8(\mu + \nu)} \left\{ r^2 + \frac{\mu}{\nu} 4\lambda^2 \left[\left(\frac{\nu}{\mu} \right)^2 \left(\frac{r}{R} \right)^2 + \frac{I_2 \left(\frac{r}{\lambda} \right)}{I_2 \left(\frac{R}{\lambda} \right)} \right] \right\} \sin 2\theta \quad (29)$$

$$\eta = \frac{T}{8(\mu + \nu)} \left\{ r^2 - 4\lambda^2 \left[\left(\frac{\nu}{\mu} \right)^2 \left(\frac{r}{R} \right)^2 + \frac{I_2 \left(\frac{r}{\lambda} \right)}{I_2 \left(\frac{R}{\lambda} \right)} \right] \right\} \sin 2\theta. \quad (30)$$

Each expression in Eq. (29) and (30) consists of two terms. Because the first terms are the same for both ϕ and η , they contribute equally to the displacement in the intracellular and extracellular spaces, so they do not contribute to the difference between the two displacements. In other words, they generate no membrane force. One could call these the “monodomain” terms, because they are the expressions one would get if the bidomain (or biphasic) nature of the tissue were ignored, and the intracellular and extracellular spaces were lumped into a single material.

The second term is proportional to λ^2 , so it will be relatively small compared to the first term. However, the expressions in the intracellular and extracellular spaces are not identical, and in fact have opposite signs, and therefore give rise to a membrane force (these are “bidomain” terms). The first term inside the bracket depends on r^2 , so it is simply a small bidomain correction to the larger monodomain behavior. The second term inside the bracket contains the Bessel function, and is therefore the boundary layer term. It is very small throughout the tissue except at the boundary, where it produces a significant membrane force.

The θ -component of the displacement is found from the radial derivative of the stream function. Because the stream function changes rapidly in the boundary layer, the contribution to u_θ and w_θ is large there. The r -component of the displacement depends on the θ derivative. Because the stream function changes gradually in the θ direction, the radial component of the displacement is small.

Figure 1 shows the intracellular displacement throughout the tissue. In this calculation, $\mu = \nu$ and $\lambda/R = 0.1$. The tissue sheet is squeezed along the fibers, and bulges outward perpendicular to the fibers. The displacement vectors form closed loops, as they must in an incompressible tissue. Because the contribution of the monodomain terms is much larger than the bidomain terms, only the monodomain behavior is readily apparent in Fig. 1, the extracellular displacement is almost identical to the intracellular displacement, and boundary layer effects are barely visible.

Figure 2 shows the difference between the intracellular and extracellular displacement, which is proportional to the membrane force. In this case, the monodomain contribution vanishes, revealing the bidomain behavior. The membrane force is large only near the boundary, where it has a characteristic four-fold symmetric appearance. Note that the arrow lengths are scaled differently in Figs. 1 and 2; the arrows at the tissue edge in Fig. 2 would be two-and-a-half times smaller than those in Fig. 1 if they had been scaled the same in both figures. If K were larger, then λ would be smaller than $0.1R$, the boundary layer at the tissue surface would be even thinner than shown in Fig. 2, and the relative scaling of the vectors in Figs. 1 and 2 would be more dramatic. However, the membrane force is $K(\mathbf{u}-\mathbf{w})$, and the leading factor of K implies that the membrane force at the boundary increases in magnitude as K increases.

The pressures p and q are not equal, and the terms proportional to D (the “bidomain” terms in Eqs. (19) and (20)) have opposite signs in the intracellular and extracellular spaces. The “monodomain” pressure is $p+q$, which is simply equal to a constant, $T/2$. Figure 3 shows the distribution of p , and Fig. 4 shows q . Interestingly, the pressure is negative in some regions of the extracellular space. The implications of having a different pressure in the intracellular and extracellular space were discussed previously (Puwal and Roth, 2010; Punal and Roth, 2012; Roth, 2013). If such a pressure difference were present for a long time, it may cause fluid to move from one space to the other.

4. Discussion

The bidomain mechanical model is a mixture or “biphasic” model that accounts for the intracellular and extracellular spaces (or phases) individually. The form of the model is similar to biphasic models developed to describe cartilage (Ateshian et al., 1997). My primary conclusion is that the mechanical bidomain model predicts that membrane forces may be large near the tissue boundary, and fall off with distance from the boundary with a length constant λ (Eq. (21)). This result is not predicted by a monodomain (or single phase) model. If membrane forces are responsible for phenomena such as mechanotransduction and remodeling, then these events may be particularly large at the tissue edge. The intracellular and extracellular displacements are nearly the same throughout the tissue. Both spaces have large strains, but because these strains are the same no membrane force develops. Only near the boundary do the intracellular and extracellular displacements differ and a membrane force exists.

The difference between the bidomain and monodomain models can be illustrated by considering stretch-induced ion channels. One hypothesis is that these channels open in response to the intracellular strain along the fiber axis (Knoll, 2002), $\epsilon_{iXX} = \epsilon_{iTT} \cos 2\theta - \epsilon_{i\theta\theta}$

$\sin 2\theta$ (Latimer et al., 2003). In my calculation, the monodomain term makes a contribution to ϵ_{xx} that is uniform throughout the tissue sheet. An alternative hypothesis is that membrane forces cause ion channels to open, in which case the bidomain model suggests that the ion channels open in the thin boundary layer at the tissue edge. This dramatic difference between the monodomain and bidomain models provides a specific prediction that can be tested experimentally.

Any mathematical model that is elementary enough to solve analytically requires simplifying assumptions. In my calculation, the model is limited to two dimensions and is based on straight, uniform fibers and a constant tension. One implication of this geometry is that the fibers approach the tissue edge at an angle, which was shown previously to highlight bidomain effects (Puwal and Roth, 2010). I assume a linear expression for the strain, which is accurate for small strains but would not hold for a piece of normal cardiac tissue undergoing contraction (Fung, 1981). This linear model enables me to obtain an analytical solution, which provides valuable insight and highlights new features of the model. However, when comparing these results to experiment, a nonlinear model would be far superior. In my example, I take the intracellular and extracellular shear moduli to be equal ($\mu = \nu$). This is most likely untrue, but one advantage of an analytical solution such as in Eqs. (29)–(30) is that you can tell by inspection how the behavior changes with different values of μ/ν . I assume that the coupling of the two spaces is described by a Hookean spring with spring constant K , but more complicated coupling could easily be imagined. The correct value for K is unknown (although my calculations suggest that K might be determined by measuring the thickness of the boundary layer), and if K is too large the boundary layer might be so thin that the macroscopic assumption of a continuum tissue averaged over many cells could become invalid. To obtain an analytical solution, I set the Young's modulus along the fibers to zero, making the intracellular shear stress/strain relationship isotropic. This assumption, which is likely invalid, could be eliminated using numerical methods. In the problem considered in this analysis, if the boundary condition were zero displacement rather than zero stress at the tissue surface, the solution would be the trivial one, zero displacement in both spaces. However, other problems exist in which a boundary layer may develop along a zero displacement boundary (Roth, 2012) as well as a zero stress boundary. Other assumptions include mechanical equilibrium (I ignore acoustic wave propagation), plane strain, and incompressibility in both spaces.

Despite these many assumptions, the analysis is useful because it makes a definite, testable prediction about where membrane forces are largest: at a free boundary. This is a *qualitatively new prediction* of the bidomain model that is not predicted by monodomain models. The boundary layer arises because of a redistribution of stresses in the intracellular and extracellular spaces in order to make the normal stress in both spaces vanish individually. The model also provides an analytical solution against which future numerical methods for solving the equations of the mechanical bidomain model can be tested. Finally, one advantage of an analytical solution is that it highlights physical behavior, such as the separation of the solution into monodomain and bidomain parts, and the relationship between the width of the boundary layer and the as-yet unmeasured tissue parameter K . Ultimately the utility of the mechanical bidomain model depends on the validity of the hypothesis that membrane forces are responsible for phenomena such as mechanotransduction and remodeling. This question can only be answered experimentally.

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Figure 1.

The intracellular displacement $\mathbf{u}$ as a function of position. The solid circle shows the tissue boundary with zero displacement, and the dashed oval shows how the tissue deforms when an active tension is present. The fibers are horizontal. The displacement arrows are scaled to make them easier to see.

Figure 2.

The difference between the intracellular and extracellular displacement, $\mathbf{u}-\mathbf{w}$, which is proportional to the membrane force. The fibers are horizontal. The arrows have been scaled by a factor of approximately 2.5 to make them easier to see; without this scaling, the arrows would be smaller than those in Fig. 1.

Figure 3. The scaled intracellular pressure, p/T , as a function of position. The fibers are horizontal. The average value is 0.5, and the pressure is positive throughout the tissue sheet.

Figure 4. The scaled extracellular pressure, q/T , as a function of position. The fibers are horizontal. The average value is zero, and the pressure is negative on the right and left, and positive on the top and bottom.