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Monodomain shear wave propagation and bidomain shear wave dispersion in an elastic model of cardiac tissue

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Abstract

Cardiac tissue elastically deforms under an applied stress, permitting shear waves to propagate through the heart. Traditionally, this behavior has been modeled with a monodomain approach, in which the mechanical properties of the intracellular and extracellular spaces are averaged together. We consider a mechanical bidomain model of cardiac tissue in which the mechanics of the intracellular and extracellular spaces are considered individually with the two spaces coupled by a spring constant. We find two normal modes of oscillation: one in which the intracellular and extracellular spaces oscillate together (a monodomain mode), and the other in which they oscillate in opposition (a bidomain mode). These two modes have unique dispersion relationships. In the extreme approximation of equal shear moduli and mass densities of the intracellular and extracellular spaces, the dispersion in the bidomain mode depends on the spring constant, while it does not in the monodomain mode.

Introduction

We have previously proposed a mechanical model [1] of heart tissue in which the elastic moduli of the actin-myosin dominated intracellular space (or phase) differs from the collagen dominated extracellular space. Weak and covalent interactions of the extracellular collagen mesh proteins are of a different bond strength than the cross-linking of the intracellular actin-myosin complex, and the collagen mesh tends to be much more isotropic in its elastic response. Prior to our discussion, others have considered elastic stresses of an ellipsoidal inclusion body [2] (such a model may be generalized to a discrete bidomain model by considering a latticework of cells as a latticework of inclusion bodies) or have proposed mechanical models of cardiac tissue in which elastic moduli are the spatially averaged response of many cells [3,4] and which do not consider the intracellular and extracellular spaces separately. Here, we derive the dispersion relation for shear waves that propagate in the intracellular and extracellular spaces of a bidomain tissue [5]. We find a non-dispersive monodomain wave in which the intracellular and extracellular waves propagate in unison, and a dispersive bidomain wave in which the intracellular and extracellular waves propagate in opposition. The dispersive bidomain wave permits us to characterize the nature of the elastic coupling force of the two spaces (e.g. whether it is proportional to the displacement difference, as we originally hypothesized) and to measure the magnitude of that coupling (e.g. the magnitude of the spring constant in our original model). We conclude that characterization of elastic wave propagation in cardiac muscle has the potential to serve as the basis for imaging the tissue – particularly ischemic tissue that has markedly different elastic response than normal tissue. Measurement of shear wave propagation in biological tissue draws upon well-known techniques [6,7,8,9] (including magnetic resonance imaging), where shear waves may be generated ultrasonically [10].

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Methods and Results

In our bidomain model of elastic tissue [1] the displacement field of the intracellular space $\vec{u}(\vec{x}, t)$ and the displacement field of the extracellular space $\vec{w}(\vec{x}, t)$ support the propagation of shear waves $\nabla \times \vec{u}$ and $\nabla \times \vec{w}$, respectively. Central to our bidomain model of elastic behavior is the idea that the intracellular and extracellular spaces are not isolated from each other – that displacements in one space can lead to displacements in the other. For example, intercalated disks are membrane structures that bridge the intracellular spaces of neighboring cells in cardiac tissue creating a continuous intracellular space called a syncytium. Intracellular displacements that stress the bridge of the intercalated disk can be translated into a stress in the extracellular space. Additionally, structures such as transmembrane extensions of the cytoskeleton can translate intracellular displacement into extracellular stress (see, for example, [11]), and *vice versa*. As a first approximation, we considered that the magnitude of this coupling force was proportional to the displacement difference between the intracellular and extracellular spaces

$$\vec{f} \sim K(\vec{u} - \vec{w}). \quad (1)$$

If the intracellular space of mass-density ρ has hydrostatic pressure p while the extracellular space of mass-density φ has hydrostatic pressure q , then the wave equations for the two spaces are derived from our original model [1,12] to be

$$-\frac{\partial p}{\partial x} + \mu \left(\frac{\partial^2 u_x}{\partial x^2} + \frac{\partial^2 u_x}{\partial y^2} \right) = K(u_x - w_x) + \rho \frac{\partial^2 u_x}{\partial t^2} \quad (2)$$

$$-\frac{\partial p}{\partial y} + \mu \left(\frac{\partial^2 u_y}{\partial x^2} + \frac{\partial^2 u_y}{\partial y^2} \right) = K(u_y - w_y) + \rho \frac{\partial^2 u_y}{\partial t^2} \quad (3)$$

$$-\frac{\partial q}{\partial x} + G \left(\frac{\partial^2 w_x}{\partial x^2} + \frac{\partial^2 w_x}{\partial y^2} \right) = -K(u_x - w_x) + \varphi \frac{\partial^2 w_x}{\partial t^2} \quad (4)$$

$$-\frac{\partial q}{\partial y} + G \left(\frac{\partial^2 w_y}{\partial x^2} + \frac{\partial^2 w_y}{\partial y^2} \right) = -K(u_y - w_y) + \varphi \frac{\partial^2 w_y}{\partial t^2} \quad (5)$$

where μ and G are the shear moduli of the intracellular and extracellular spaces, respectively. (Note that Eqs. 2–5 are derived under the assumption of incompressible intracellular and extracellular spaces, where $\nabla \cdot \vec{u} = 0$ and $\nabla \cdot \vec{w} = 0$.) While in cardiac tissue the intracellular space is typically more anisotropic than the extracellular space and can generate active tension, for this study we represent the intracellular and extracellular spaces as isotropic and passive with differing shear moduli. By taking the appropriate derivative and combining the above relations, we may eliminate consideration of the hydrostatic pressures and obtain the relations

$$\mu \nabla^2 (\nabla \times \vec{u}) = K(\nabla \times \vec{u} - \nabla \times \vec{w}) + \rho \frac{\partial^2}{\partial t^2} (\nabla \times \vec{u}) \quad (6)$$

$$G\nabla^2(\nabla \times \vec{w}) = -K(\nabla \times \vec{u} - \nabla \times \vec{w}) + \varphi \frac{\partial^2}{\partial t^2}(\nabla \times \vec{w}) \quad (7)$$

where $\nabla = \hat{x} \frac{\partial}{\partial x} + \hat{y} \frac{\partial}{\partial y}$ is the del/nabla operator and $\hat{x}$ and $\hat{y}$ are the usual Cartesian unit vectors [13]. With both spaces isotropic we are free to define the x -direction as the direction of propagation with oscillations in the z -direction; the shear wave trial solutions [13] are

$$\nabla \times \vec{u} = u_0 e^{i(kx - \omega t)} \hat{z} \quad (8)$$

$$\nabla \times \vec{w} = w_0 e^{i(kx - \omega t)} \hat{z} \quad (9)$$

where k and ω are the spatial and angular frequencies (k should not be confused with the coupling force spring constant K); the relation between amplitudes is given by

$$\frac{u_0}{w_0} = \frac{k^2 \left(\frac{\mu\varphi}{\rho} - G \right) + K \left(1 + \frac{\varphi}{\rho} \right) \pm \varphi R}{k^2 \left(\mu - \frac{G\rho}{\varphi} \right) - K \left(1 + \frac{\varphi}{\rho} \right) \mp \rho R} \quad (10)$$

where R is the root

$$R = \sqrt{\frac{4K^2}{\varphi\rho} + \left(\left(\frac{G}{\varphi} - \frac{\mu}{\rho} \right) k^2 + \left(\frac{1}{\varphi} - \frac{1}{\rho} \right) K \right)^2}. \quad (11)$$

The dispersion relation between the angular frequency and the spatial frequency is found to be

$$\omega^2 = \frac{1}{2} \left(\left(\frac{G}{\varphi} + \frac{\mu}{\rho} \right) k^2 + \left(\frac{1}{\varphi} + \frac{1}{\rho} \right) K \right) \pm \frac{R}{2} \quad (12)$$

We note that the $\pm$ and $\mp$ occurring in Eqs. 10 and 12 are because we have assumed a positive square root in Eq. 11. The results are, perhaps, more illuminating if we make the assumption that the intracellular and extracellular spaces have equal densities ($\varphi = \rho$) and equal shear moduli ($G = \mu$). In this case the monodomain solution (subtraction in the numerator and addition in the denominator of Eq. 10) yields a relation between amplitudes

$$\frac{u_0}{w_0} = 1 \quad (13)$$

and a dispersion relation (subtraction in Eq. 12)

$$\omega^2 = \frac{\mu}{\rho} k^2 \quad (14)$$

and, thus, a group velocity equal to the phase velocity

$$v_{group} = v_{phase} = \sqrt{\frac{\mu}{\rho}}. \quad (15)$$

Meanwhile, the bidomain solution (addition in the numerator and subtraction in the denominator of Eq. 10) yields a relation between amplitudes

$$\frac{u_0}{w_0} = -1 \quad (16)$$

and a dispersion relation (addition in Eq. 12)

$$\omega^2 = \frac{\mu}{\rho} k^2 + \frac{2K}{\rho}. \quad (17)$$

Here, we clearly have a dispersive wave previously not predicted by monodomain considerations in which the group velocity ω/k [13] is still

$$v_{group} = \sqrt{\frac{\mu}{\rho}} \quad (18)$$

while the phase velocity ω/k [13] is

$$v_{phase} = \sqrt{\frac{\mu}{\rho} + \frac{2K}{\rho k^2}}. \quad (19)$$

Discussion

In our original presentation of a mechanical model of elastic heart tissue [1], we considered that the intracellular and extracellular spaces exhibit unequal elasticity; in other words that heart tissue exhibited mechanical bidomain behavior in addition to its well known electrical bidomain behavior. We further considered, as a first approximation, that the coupling force of the intracellular and extracellular spaces was linearly proportional to the displacement difference of those two spaces [1,14]. This hypothesis was made *a priori* without experimental evidence. However, this bidomain model predicts a new type of shear wave (never previously analyzed) in which the intracellular and extracellular spaces oscillate in opposition; when we consider that the prominent bidomain effect is dispersive and that the dispersion is directly related to the coupling spring constant (Eqs. 17 and 19), we are provided with a means of validating that hypothesis and measuring the magnitude of the spring constant. The previously known monodomain model of tissue admits wave solutions similar to the acoustic mode of phonon propagation, while this new bidomain mode is analogous to the optical mode of phonon propagation [15]. A more fundamental analogy may be found with the system of two coupled oscillators [16], such as two pendulums coupled by a spring. This system has two eigenmodes – a low frequency mode in which the oscillators are in phase, and a high frequency mode in which the oscillators are out of phase.

Our analysis, here, is limited to shear wave propagation because we consider the intracellular and extracellular spaces to be incompressible and, hence, not admit pressure waves [1,12,14]. This assumption of incompressibility was based on the assumption that a large fraction of the tissue is water and that water is highly incompressible. A more complete analysis of tissue would consider compressibility and, hence, admit pressure waves for consideration. Moreover, we stress applications of this model to cardiac tissue, but the model is applicable to other tissues.

In summary, a more complete description of cardiac tissue considers bidomain continua and recognizes that these spaces are coupled [17]. Our preliminary result is the prediction of a new type of shear wave associated with the bidomain model, which is dispersive and which has the intracellular and extracellular spaces moving in opposition. Through analysis of admitted wave solutions we may confirm this hypothesis, and we are provided with a potential imaging technique (a macroscopic tool) for measuring the elastic coupling of the intracellular and extracellular spaces (a microscopic parameter).

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