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A Mathematical Framework for Predicting the Location and Extent of Complex Cardiac Ruptures

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Cardiac rupture after myocardial infarction is a high-mortality complication whose spatial pattern remains poorly predicted [1]. This note argues that the appropriate mathematical framework is anisotropic computational fracture mechanics, specifically the coupling of finite element analysis with a crack phase-field model that incorporates myocardial fiber orientation [2]. The clinical observation that dissection paths originate where the components of the helical ventricular myocardial band converge suggests a geometric and material discontinuity, which we formalize as a weak interface in a fibrous continuum [1]. We identify the mathematical objects involved: an anisotropic hyperelastic constitutive law, a phase-field variable governed by an energy-based anisotropic criterion, and a finite element discretization that resolves the fiber field from diffusion tensor imaging [3]. The result is a field theory of material failure that predicts a rupture surface rather than a probability, and it constitutes the natural mathematical extension of the author's clinical research line.

Keywords: anisotropic fracture; crack phase-field; cardiac rupture; finite element analysis; myocardial fiber architecture; intramyocardial dissection.

1 The mathematical object

The clinical problem is stated geometrically: given a ventricular domain $\Omega \subset \mathbb{R}^3$ with a known infarct region $\Omega_{\text{inf}} \subset \Omega$, predict the initiation site and propagation path of a rupture [1]. The relevant mathematical object is not a scalar risk score but a *vector field of material failure*, defined on the same domain that carries the stress state.

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The passive myocardium is modeled as a fiber-reinforced hyperelastic continuum. Let $\mathbf{F}$ be the deformation gradient and $\mathbf{C} = \mathbf{F}^{\top}\mathbf{F}$ the right Cauchy–Green tensor. The strain energy is decomposed as

$$\Psi = \Psi_{\text{iso}}(\mathbf{C}) + \sum_{i=\text{f,s}} \Psi_{\text{aniso}}(\mathbf{C}, \mathbf{a}_i \otimes \mathbf{a}_i), \tag{1}$$

where $\mathbf{a}_{\text{f}}$ and $\mathbf{a}_{\text{s}}$ are the fiber and sheetlet directions obtained from diffusion tensor imaging tractography [3]. This is the standard constitutive foundation for cardiac finite element models [4].

The rupture itself is described by a *phase-field variable* $d : \Omega \to [0, 1]$, where $d = 0$ denotes intact tissue and $d = 1$ fully fractured material. The total energy functional is

$$\mathcal{E}(\mathbf{u}, d) = \int_{\Omega} \Psi(\mathbf{C})\, \mathrm{d}V + \int_{\Omega} G_c\, \gamma(d, \nabla d)\, \mathrm{d}V, \tag{2}$$

where G_c is the fracture toughness and γ is the crack surface density function [2]. The critical point of (2) yields the coupled displacement–damage problem.

2 The anisotropic criterion

The essential mathematical ingredient for this specific clinical problem is *anisotropy of the fracture criterion*. In an isotropic phase-field model, the crack path is determined solely by the stress field. In the myocardium, the fiber architecture biases propagation: a crack oriented parallel to the fibers requires less energy than one crossing them [2].

The energy-based anisotropic criterion replaces the scalar G_c by a directional fracture toughness $G_c(\mathbf{n})$, where $\mathbf{n}$ is the crack normal. A tractable form is

$$G_c(\mathbf{n}) = G_{c,\perp} + (G_{c,\parallel} - G_{c,\perp})\, (\mathbf{n} \cdot \mathbf{a}_{\text{f}})^2, \tag{3}$$

with $G_{c,\parallel} < G_{c,\perp}$ reflecting the experimentally observed preference for fiber-parallel splitting [2]. Substituting (3) into (2) yields a phase-field evolution equation whose minimizers are crack paths that align with the local fiber direction.

This is precisely the mathematical mechanism required to formalize the clinical hypothesis: the "convergence of three components" described in the clinical report of Vargas et al. [1] corresponds to a region where $\mathbf{a}_{\text{f}}$ rotates rapidly, so that the directional toughness (3) varies steeply in space. Within this formulation, such a region is expected to act as a *geometric attractor* for the phase-field: because $G_c(\mathbf{n})$ varies steeply there, the phase-field evolution is biased toward concentrating damage and initiating the dissection path along that interface. In this note, *dissection path* and *rupture surface* are used interchangeably: the phase-field level set $d = 1$ is a two-dimensional surface in Ω, whose intersection with the endocardium traces the clinically observed dissection path.

3 The computational realization

The anisotropic criterion (3) is discretized together with (2) as follows. The displacement field $\mathbf{u}$ and the phase-field d are approximated on a mesh $\mathcal{T}_h$ of the ventricular geometry, reconstructed from magnetic resonance imaging. The fiber directions $\mathbf{a}_{\text{f}}$ are interpolated from diffusion tensor tractography data [3], which provides the spatial field of myocardial architecture at resolutions of the order of a few hundred micrometers—sufficient to resolve the transmural fiber rotation.

The coupled system is solved incrementally under ventricular pressure loading, with the infarct region assigned degraded material properties consistent with scar tissue [4]. The output is a spatiotemporal field $d(\mathbf{x}, t)$ whose level sets trace the predicted dissection surface.

A historical precedent supports this choice: Reif and Silver [4] used a finite element model of the infarcted left ventricle to show that maximum von Mises stress concentrates at the endocardial surface near the infarct border, and proposed the pathogenic sequence *stress concentration* → *endocardial tear* → *dissecting hemorrhage*. The phase-field formulation extends that static stress analysis into a predictive theory of *where the tear propagates*.

4 Outlook

The mathematical line established in this note is anisotropic crack phase-field modeling coupled with finite element analysis of the fiber-reinforced myocardium [2, 4]. This framework formalizes the clinical research line pursued by the author. Specifically, the convergence of the three components of the helical ventricular myocardial band documented in [1] corresponds to a region of rapid fiber rotation, which in the model appears as a steep spatial gradient of directional toughness $G_c(\mathbf{n})$; the dissection paths observed clinically are then predicted as minimizers of the phase-field functional in that region. The output of the framework is therefore a predicted rupture surface on the ventricular domain, not a scalar probability.

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