# A computational framework for electromechanical contact between excitable deformable cells

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***Abstract*—Excitable deformable cells act as cooperative structures and show emergent behaviors from their interactions. The theoretical modeling and numerical simulation of such an interaction is far from being fully unveiled though information transmission mismatches have been shown to underline severe pathological conditions. In this study we provide a novel contact mechanics computational framework simulating the cooperative interaction among cardiac myocytes.**

***Keywords*—Cardiomyocytes modeling, Active-strain, Contact Mechanics, Nonlinear coupled PDEs.**

## I. Introduction

CARDIAC tissue is a complex heterogeneous and anisotropic medium characterized by nonlinear and multiscale dynamics. Imperfect mechanotransduction in the cardiac tissue, converting mechanical deformation into a change in cell growth or remodeling, is a cutting edge research topic [1]-[2] since it can lead to a variety of diseases. In order to describe the dominant mechanisms occurring at different scales within a constitutive framework for the cardiac tissue, microstructural properties have to be properly described, including mechano-regulated interactions occurring among the tissue constituents. In particular, modeling and simulation of contact interactions between myocytes is a topic still unchallenged today and a progress in this field would allow the study of emergent phenomena considered at the forefront of research in cardiovascular biomechanics. In this perspective, in the present contribution, a novel constitutive and computational framework for the simulation of contact between cardiac myocytes is proposed. The present work introduces important novelties with respect to the current state-of-the-art. First, we extend the single cell study proposed in [3], by formulating a novel constitutive model for electro-mechanical contact between myocytes. Second, we provide a novel finite element procedure introducing a new structure-structure interaction with mixed type boundary conditions, for two-dimensional bodies with nonlinear electro-mechanical coupling. A representative example of the problem is provided in figure.

## II. Continuum model of the active-strain myocyte

The kinematics of myocytes is framed within the classical description of continuum mechanics under finite elasticity assumptions and specialized for two-dimensional domains. In order to encompass the nonlinear coupling between the electrophysiological dynamics and the hyperelastic material response induced by the excitation-contraction mechanisms along the fibers directions in a single cardiomyocyte, the multiplicative decomposition of the deformation gradient into an elastic (passive) part and an active part is assumed [3]-[4]-[5] The mechanical model is based on the active-strain formulation [3]. The activation variables dynamics, which are responsible for the contraction and thickening of the medium, are ruled by the two-variable phenomenological Rogers-McCulloch's model [8]. The model has been demonstrated to capture the main features of the action potential spatio-temporal dynamics with a reduced mathematical complexity though several possible extensions are available in the literature [9]. The arising nonlinear coupled system of equations, describing the atctive-strain elctromechanical dynamics of a single cell, is solved numerically using a finite element procedure. Following previous studies [10], a possible solution strategy is based on staggered schemes for the solution of the coupled system. This procedure consists in dividing the problem into a mechanical phase and an electrophysiological phase, corresponding to the equations and solving at each time step the mechanical problem first and then the electrophysiological problem. Both the mechanical and the electrophysiological model are nonlinear systems requiring a nested Newton-Raphson iterative scheme

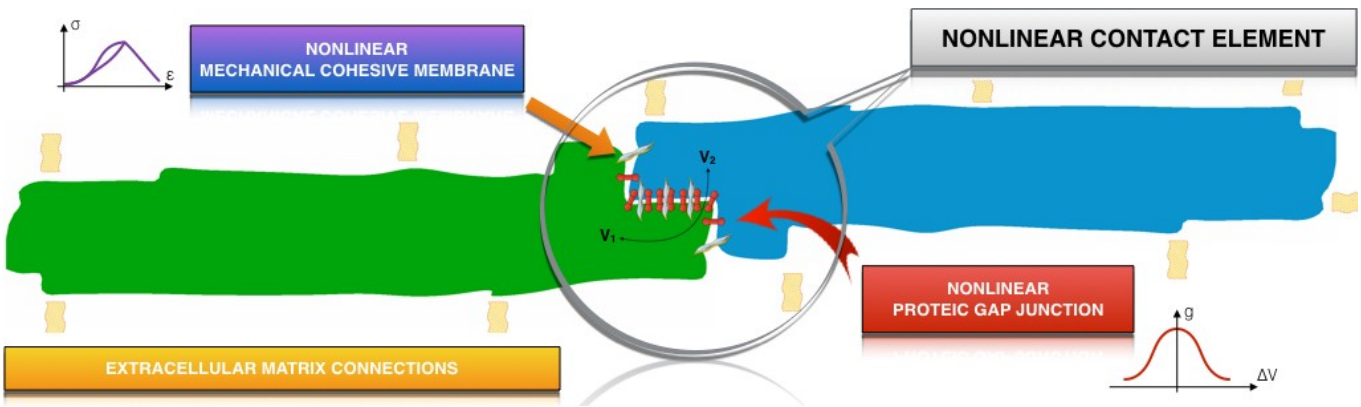


**Fig. 1** Representative scheme of cell-cell multiphysics contact mechanics.


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for the numerical solution.

## III. Structure-structure electromechanical interaction model

The exchange of the electrical impulse between two cells is ruled by clusters of intercellular proteic channels, namely gap junctions [11], that are voltage dependent and can induce important effects on the overall emerging dynamics. Imperfect mechanotransduction in the cardiac tissue, converting mechanical deformation into a change in cell growth or remodeling, is a cutting edge research topic, since it can lead to a variety of diseases. In order to encompass the complex nonlinear cell-cell interaction mechanism it is introduced a set of internal boundary conditions defined on the contact boundary between the two myocytes.

As far as the mechanical response is concerned, tractions exchanged at the contact boundary must be continuous for equilibrium considerations and are function of the relative displacements. Experimental results based on atomic force microscope measurements clearly pinpoint the existence of a contact regime [1], which can be modelled as a traction-separation constitutive relation [12]-[13] of the form:

$$T_n = T_{n,max}\, g_n / g_{n,max}, if\ 0 < g_n < g_{n,max},$$

and $T_n = 0, if\ g_n > g_{n,max}$,

where $T_n$ is the normal component of the cohesive traction vector at the contact boundary, and $g_n$ is the normal relative opening displacement, while $g_{n,max}$ $T_{n,max}$ are respectively critical gap opening and maximum value of the normal traction.

. As far as the transfer of electric signals across the contact boundary is concerned, it is assumed that the current flows solely in the direction normal to the interface. According to the experimental evidence [11], the contact diffusivity at the boundary $D_n$ is modeled as a nonlinear function of the transjunctional voltage gap of the form:

$$D_n = a_1 + a_2 \left( \frac{1}{1+e^{-a_1 - a_4 \Delta v}} - \frac{1}{1+e^{-a_1 + a_4 \Delta v}} \right),$$

where $\Delta v$ is the transjunctional gap between the two myocytes and the other constants are model parameters.

## IV. Conclusion

The proposed novel constitutive model, validated against experimental evidences, reproduces static and dynamic observed behaviors with particular attention to (i) length and slope of contact boundary, (ii) maximum tractions at the interface, (iii) cell width-length ratio, and (iv) propagation rate of action potential between adjacent myocytes. The proposed computational contact framework opens a new micro-scale level of analysis within the context of nonlinear interaction among biological objects. Forthcoming contributions will target different biomedical applications, e.g., crosstalk-based cardiovascular diseases, low energy defibrillation devices, and microfluidic chip design and optimization.


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