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A computational model of the left ventricle biomechanics using a composite material approach

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ABSTRACT

Several computational models are available for studying cardiac mechanics. These models incorporate tissue passive/active response in conjunction with hyperelasticity and anisotropy. For capturing the active response they involve complicated non-conventional strain energy functions which account for tissue active contraction. They are either implemented as custom-developed non-linear finite element (FE) codes or require user-defined subroutines compiled within commercial FE software packages. Difficulty of computational implementation of such models remains an issue for the research community. Furthermore, myocardial tissue has sophisticated microstructure while pathologies may alter its constituents and their organization. Hence, cardiac mechanics models adaptable to various pathological conditions are advantageous. This paper aims at developing a cardiac mechanics model using a novel approach which does not require strain energy functions developed specifically for simulating myocardial tissue active response, lending itself for effective implementation in off-the-shelf FE solvers. This model considers myocardial hyperelasticity, anisotropy, and active contraction. It was developed using a tissue composite material model which includes two major parts: background part and myofibers. The model was applied to an *in silico* geometry of a canine left ventricle (LV). Resulting diastolic pressure-volume curve shows very good agreement with corresponding experimental observations. Also, calculated mid-ventricular end-diastolic strain components are within one standard deviation of measurements performed through the LV equatorial area. Furthermore, computed end-systolic strain components and ejection fraction are close to or within one standard deviation of in-vivo measurements of a beating canine LV. These results demonstrate that the proposed model can be employed as an effective alternative for studying cardiac mechanics.

1. Introduction

Computational models of cardiac mechanics can be utilized as effective tools in diagnosis and patient-specific therapy planning (Augenstein, Cowan, LeGrice, & Young, 2006; Hassan, Hamdi, & Noma, 2012; Marchesseau et al., 2013). These models can assist in classifying myocardial pathologies, adopting therapeutic procedures, and even predicting therapy outcomes

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(Drzewiecki, Wang, Li, Kedem, & Weiss, 1996; Goktepe & Kuhl, 2010; Marchesseau et al., 2013). The heart function is similar to that of a pump where pathological conditions are analogous to mechanical issues that alter its efficiency. These alterations can be quantified in terms of parameters such as ejection fraction (EF), displacement, strain, and stress fields. For instance, prolonged hypertension yields elevation of end-systolic wall stress and depression of fractional fiber shortening, leading to thick-walled hypertrophic ventricles, which is a cardiac physiological response to excessive workload (Christy, 2012). Cardiac ischemia is another prevalent coronary artery disease which may be followed by myocardial infarction (MI) in acute cases. Infarcted scar leads to immediate abnormal mechanical response (e.g. weaker contraction compared to normal tissue) (Lee et al., 2014; Sermesant, Delingette, & Ayache, 2004; Veress, Segars, Tsui, & Gullberg, 2011). It is subsequently replaced by stiffer fibrous tissue following a fibrosis phase. Stiffer collagenous scar tissue remains after fibrosis which amplifies mechanical stress in the infarct region, leading to irregular stretching patterns within the myocardium (Sermesant et al., 2004; Veress et al., 2011). Accordingly, such irregular patterns can be assessed based on reliable cardiac mechanics models, potentially leading to diagnostic information. Over the past few decades, diverse computational models of cardiac mechanics were developed based on various assumptions characterizing cardiac tissue mechanics. While linearity and isotropy have been utilized as simplifying assumptions for myocardial modeling (Cupps et al., 2003; Hu et al., 2003; Nehorai & Jeremic, 1998), it is well-known that cardiac tissue can be accurately characterized by anisotropic hyperelastic models (Choi, D' hooge, Rademakers, & Claus, 2010; Costa et al., 1996; Wang et al., 2013). The majority of hyperelastic anisotropic models postulate a constitutive law in terms of the tissue strain energy (Bovendeerd, Arts, Huyghe, Van Campen, & Reneman, 1992; Costa, Holmes, & Mcculloch, 2001; Dal, Goktepe, Kaliske, & Kuhl, 2013; Goktepe, Acharya, Wong, & Kuhl, 2011; Keldermann, Nash, Gelderblom, Wang, & Panfilov, 2010). While efficient, such models may not be easily adaptable for pathologies associated with substantial alterations in tissue microstructure and their corresponding properties (Agnoletti et al., 2006; Ertl & Frantz, 2005; Holmes, Borg, & Covell, 2005; Hubbard & Henriquez, 2012; Katz, 2010). Recent models consider different constitutive laws for pathological cardiac tissue. For instance, Sun et al. (2009) and Wenk, Sun et al. (2011) assign different constitutive laws to regions suffering from a left ventricular (LV) aneurysm. With advances in cardiac tissue engineering and biomaterial therapies, pathological portions of the heart may be replaced by biomaterials with different mechanical properties to improve cardiac mechanical function and prevent further remodeling (Gálvez-Montón, Prat-Vidal, Roura, Soler-Botija, & Bayes-Genis, 2013; Wenk, Eslami et al., 2011; Zhang et al., 2005). Hence, it is desirable to have cardiac mechanics models adaptable to diverse pathological and therapeutic conditions by locally assigning altered mechanical properties based on existing knowledge of corresponding tissue microstructure alteration. In addition to its microstructural complexity and variability under normal and pathological conditions, cardiac tissue is active as it generates contraction during the systole phase, elevating cardiac tissue mechanics complexity to a higher level. Current LV mechanical models idealize the LV tissue as homogeneous, anisotropic and hyperelastic material. To account for the LV muscle's anisotropy, hyperelasticity and most importantly active contraction generation, they use highly complex constitutive models. Incorporating such constitutive models within FEM framework has led to custom developed FE solvers (Bovendeerd et al., 1992; Choi et al., 2010; Christy, 2012; Costa et al., 1996; Costa et al., 2001; Cupps et al., 2003; Goktepe et al., 2011; Hu et al., 2003; Keldermann et al., 2010; Lee et al., 2014; Nehorai & Jeremic, 1998; Sermesant et al., 2004; Veress et al., 2011; Wang et al., 2013), which are not easily adaptable for implementation in commercial FE solvers (Dal et al., 2013; Goktepe & Kuhl, 2010), hence lending very limited availability to the cardiac mechanics research community. Recently, efforts were made to implement realistic diastolic and systolic cardiac mechanics models using available off-the-shelf FE solvers (Genet et al., 2014; Genet, Lee, Baillargeon, Guccione, & Kuhl, 2015). However, such implementations still require user-defined subroutines which are not available for the research community. FEBio is also a free FE solver recently developed for biomechanical applications including LV mechanical modeling. This solver's development is ongoing and while it is expected to be an effective tool in the future, its current version only provides limited options to model LV tissue anisotropy and hyperelasticity (Maas, Ellis, Ateshian, & Weiss, 2012). For patient-specific fiber orientation distribution with arbitrary hyperelastic model, user-defined subroutines are required (Maas, Rawlins, Weiss, & Ateshian, 2015). Here, we propose an LV mechanical model which is adaptable to normal and various pathological LV contraction scenarios. The model follows a novel approach to account for tissue anisotropy, hyperelasticity, and active fiber contraction forces while, unlike other relevant models which idealize the cardiac tissue as a homogeneous material in conjunction with strain energy functions developed specifically for cardiac tissue, it idealizes the tissue as a composite material which allows using conventional strain energy functions. As such, it can be implemented in commercial FE solvers without requiring user-defined subroutines, paving the way for broad involvement of the research community to study cardiac disorders using effective mechanical modeling tools. The novelty of the model lies in treating the myocardial tissue as a composite material including a background tissue through which microscopic reinforcement bars (fibers) undergoing time-variable contraction forces are distributed. The model was utilized to mimic LV diastolic and systolic mechanical behavior and its contractile performance was compared with other *in silico* models and relevant in-vivo measurements of the LV contraction.

2. Materials and methods

2.1. Composite material model

The LV contracts due to contraction force generation within cardiac fibers, leading to blood pressure alteration on its endocardial surface. In fact, cardiac contraction forces work to maintain this evolving blood pressure and to continue contrac-