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A framework for biomechanics simulations using four-chamber cardiac models



Arian Jafari, Edward Psczolkowski, Adarsh Krishnamurthy*

Mechanical Engineering Department, Iowa State University, United States

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ABSTRACT

Computational cardiac models have been extensively used to study different cardiac biomechanics; specifically, finite-element analysis has been one of the tools used to study the internal stresses and strains in the cardiac wall during the cardiac cycle. Cubic-Hermite finite element meshes have been used for simulating cardiac biomechanics due to their convergence characteristics and their ability to capture smooth geometries compactly—fewer elements are needed to build the cardiac geometry—compared to linear tetrahedral meshes. Such meshes have previously been used only with simple ventricular geometries with non-physiological boundary conditions due to challenges associated with creating cubic-Hermite meshes of the complex heart geometry. However, it is critical to accurately capture the different geometric characteristics of the heart and apply physiologically equivalent boundary conditions to replicate the *in vivo* heart motion. In this work, we created a four-chamber cardiac model utilizing cubic-Hermite elements and simulated a full cardiac cycle by coupling the 3D finite element model with a lumped circulation model. The myocardial fiber-orientations were interpolated within the mesh using the Log-Euclidean method to overcome the singularity associated with interpolation of orthogonal matrices. Physiologically equivalent rigid body constraints were applied to the nodes along the valve plane and the accuracy of the resulting simulations were validated using open source clinical data. We then simulated a complete cardiac cycle of a healthy heart and a heart with acute myocardial infarction. We compared the pumping functionality of the heart for both cases by calculating the ventricular work. We observed a 20% reduction in acute work done by the heart immediately after myocardial infarction. The myocardial wall displacements obtained from the four-chamber model are comparable to actual patient data, without requiring complicated non-physiological boundary conditions usually required in truncated ventricular heart models.

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1. Introduction:

Computational models of cardiac biomechanics can improve the fundamental understanding of the cardiovascular system by providing access to different quantities of interest that cannot be directly measured or require invasive procedures. Computational models have been used to study normal cardiac physiology (Kerckhoffs et al., 2007) and pathological conditions such as heart failure (Kerckhoffs et al., 2010; Niederer et al., 2011), myocardial infarction (Wang et al., 2011), etc. Most existing computational biomechanics cardiac models focus only on specific regions of the heart such as the left ventricle or both ventricles without

explicitly modeling the atria. The complex and irregular geometry of the left and right atria of the heart have restricted most modeling and simulation to ventricular models. Excluding parts of the heart geometry necessitates the use of non-physiological boundary conditions that require extensive and tedious tuning to match the simulated cardiac motion with patient data. Advances in non-invasive imaging technology have made it feasible to generate patient-specific ventricular models (Aguado-Sierra et al., 2011; Krishnamurthy et al., 2013a), but it remains difficult to create high-quality meshes that include anatomic features such as valve annuli or atria automatically. A four-chamber cardiac model will enable the use of a wide variety of physiologically equivalent boundary conditions for each specific patient that can optimally match the cardiac motion with patient data.

Cubic-Hermite finite element interpolation schemes have been popular in cardiac modeling because of their convergence properties in finite element simulations of ventricular biomechanics

* Corresponding author.
E-mail addresses: ajafari@iastate.edu (A. Jafari), edwardp@iastate.edu (E. Psczolkowski), adarsh@iastate.edu (A. Krishnamurthy).

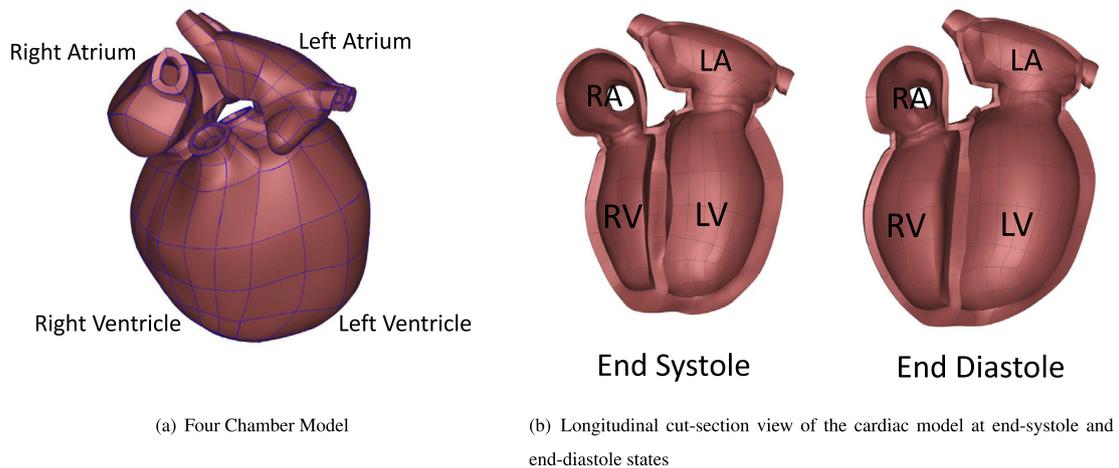


Fig. 1. Topologically complex four-chamber model and a four-chamber model with valve annuli at the end-diastolic and end-systolic states of the cardiac cycle. The left ventricle is shown on the right side due to better informative cut-section view.

(Costa et al., 1996). However, construction of cubic-Hermite geometric meshes has been limited to ventricular geometries below the valve plane due to difficulties in handling complex topologies of the atria and great veins (Fig. 1). Recently, Land and Niederer (2018) extended their biventricular geometry which was built initially using cubic-Hermite elements to a four-chamber model using the tetrahedral finite elements to overcome the more complex topology associated with atria and studied the effects of atrial contraction on whole organ function. By applying different types of boundary conditions they showed that ventricular pressure-volume curves do not change significantly. Krishnamurthy et al. (2016) made use of cubic-Hermite meshes with extraordinary nodes to model the complex geometry of the heart with valve annuli and perform biomechanics simulations. Here we extend the same extraordinary node concept to construct a four-chamber cubic-Hermite finite element model and perform full-beat simulations.

Modeling the local fiber architecture of the heart muscles is a challenge, since it can be computationally expensive or inaccurate depending on the interpolation method used. In addition, the fiber architecture of the atria is not that extensively studied as the ventricles (Gonzales et al., 2013). Detailed histological studies and diffusion tensor imaging of the ventricles show that the heart muscle fibers rotate 120° from the epicardial to the endocardial surface (Wong and Kuhl, 2014) and this rotation can be reasonably considered similar for a large group of individuals (Lombaert et al., 2011). On the other hand, there seems to be not much variation in the fiber direction through the atrial wall due to its thin structure. Among all the fiber mapping methods, using a coordinate-frame interpolation scheme with the log-Euclidean transformation guarantees smooth interpolation accounting for the shape and the size of the cardiac geometry (Krishnamurthy et al., 2013a). In this work, we make use of the cubic-Hermite mesh topology to orient the fibers in the atria and use the log-Euclidean transformation to interpolate the fibers in the ventricles.

Simulating a complete cardiac cycle requires the correct chamber pressures to be applied to the four chambers. To perform this, the 3D finite element model needs to be coupled with a circulatory model that includes both pulmonary and systemic circulations (Kerckhoffs et al., 2006). Traditionally, the circulatory system is modeled using a set of lumped-parameter Windkessel models. We make use of the CircAdapt circulation model (Arts et al., 2005) to model the circulation and couple it with the ventricles of our finite element model.

Myocardial infarction is a leading cause of heart failure that occurs due to blood blockage to some regions of the heart, causing

damage to the heart muscles. Cardiac motion can be used as a diagnostic tool to identify the effects of myocardial infarction. Cardiac simulation can help understand the acute effect of myocardial infarction on the cardiac motion. However, correctly replicating the motion of the heart after myocardial infarction requires corresponding changes to the active muscle properties in the infarcted region and modeling the effect of atrial structures of the heart on the deformations. In this work, we show that using a four-chamber model can help in assessing the acute impact of the myocardial infarction on the cardiac motion.

2. Methods

2.1. Geometric modeling of the four-chamber heart

We built the 3D atlas mesh for the current work based on a previously constructed 3D biventricular mesh model (Zhang et al., 2012) and a biatrial model (Gonzales et al., 2013), both of which are publicly available. The atria were attached to biventricular model manually by connecting the nodes along the valve annuli. This atlas mesh was then modified using the data from the literature for healthy humans. The atlas mesh was imported as a 3D obj format in Blender. Using the imported mesh in Blender the ventricular and atrial walls were adjusted by moving cubic-Hermite mesh nodes. However, we had to manually subdivide some elements, since creating a perfectly matched cubic-Hermite mesh including all four chambers leads to skewed elements around the valve annuli. Skewed elements might cause divergence during the finite element analysis, therefore, we manually inserted newer elements without deviating too much from the actual geometry. The data from literature including the average unloaded volume of the human left and right ventricles (Hudsmith et al. (2005)) were used to adjust the cardiac size after the geometry building process. The unloaded volumes are 47 ml and 49 ml for the RV and LV, respectively. The number of nodes, elements, and the degrees of freedom are tabulated in Table 1.

The model includes both the left and the right ventricles, orifices and valve annuli, and both the left and the right atria in a regular human heart. The model dimensions including the volume of both ventricles, the volume of both atria, and wall (myocardium) thickness were taken from the available literature. As stated earlier, The finite element mesh of the cardiac model was generated with an open-source 3D computer graphics software, "Blender", similar to the methods described by Krishnamurthy et al. (2015). Utilizing these user-defined tools makes geometry editing more

Table 1
Comparison between the present work and Augustin et al. (2016) of a four-chamber cardiac finite element model.

	Element type	# nodes	# elements	# DOF
Present work	Cubic-Hermite	968	480	30720
Augustin et al. (2016)	Tetrahedral	–	184.6×10^6	95.9×10^6

efficient as complex repeated functions is automated and performed simultaneously. Compared to past methods of defining geometry from patient data, which relied heavily on manually and meticulously placing individual nodes on cross-sectional cardiac images, this new method and tool-set has drastically cut down on model build time.

After generating the geometric mesh of the heart, we have to determine an unloaded reference state (the state at which the ventricle cavity pressure in a passive state is zero) to accurately compute the stresses. Dimensional data obtained from clinical images *in vivo* are not in the unloaded state due to continuous heart motion (Alastrue et al., 2008). Many researchers have tried to consider the end-systolic (Walker et al., 2005) or mid-diastolic (Sermesant and Razavi, 2010) geometry as the unloaded state, but it was shown later that the unloaded state deviates from both these states (Klotz et al., 2006). The method developed by Rajagopal et al. (2006) can be used to estimate the reference state for a wide variety of problems by using inverse methods. We had previously applied this method to compute the unloaded state for biventricular models (Krishnamurthy et al., 2013a) from the measured end-diastolic geometry, pressure, and passive material properties through an iterative method. However, in our case, we do not have a patient-specific geometry at the end-diastolic state to apply this method. Hence, we make use of the empirical formula provided by Klotz et al. (2006), which correlates the unloaded left-ventricular volume to the end-diastolic volume and pressure, to rescale the ventricular geometry to the correct volume. We also keep the ventricular wall volume constant to account for the incompressibility in the simulations.

2.2. Modeling fiber orientation

Modeling the fiber architecture of both the ventricles and the atria is important for accurately capturing the cardiac deformations. Krishnamurthy et al. (2013a) used the diffusion tensor \mathbf{D} , which is a 3×3 symmetric, positive-definite, covariance matrix representing the local voxel-averaged distribution of the diffusion of water molecules to define the local fiber coordinate system. By applying a coordinate-frame interpolation, they were able to guarantee a smooth interpolation of the fiber direction especially around extraordinary vertices (Krishnamurthy et al., 2016). However, it has been shown that the fiber orientation in the ventricles vary from -60° to $+60^\circ$ with respect to the circumferential direction. In addition, a recent statistical analysis (Lombaert et al., 2012) of fiber architecture variation in a population of human hearts has revealed that fiber orientations are well preserved between individuals. Unlike ventricles, the thin structure of the atrial wall makes it difficult to measure its fiber angles. Therefore, a fixed fiber angle for the whole atria was used in previous works, which is based qualitatively on published diagrams of atrial fiber tracts (Krueger et al., 2011; Gonzales et al., 2013). The significant changes in the ventricles' fiber angles in the transmural direction necessitate an interpolation method to model the fiber orientations accurately and smoothly.

In this work, we make use of a coordinate-frame interpolation scheme that uses the log-Euclidean transformation (see Appendix C for details). This method provides a simple way to specify the cardiac fiber orientations in complex cardiac models. We calculate

the orthogonal matrix \mathbf{F}_{endo} by calculating the circumferential, radial, and transverse direction and rotating it by $+60^\circ$ with respect to the circumferential direction and assigned to the endocardial nodes. Similarly the \mathbf{F}_{epi} is calculated and assigned to the epicardial nodes. The circumferential, radial, and transverse directions are explicitly calculated from the mesh, since the cubic Hermite elements are oriented along these directions in our model. The log-Euclidean interpolation is then used to calculate the fiber orientations at any position inside the cardiac wall. As can be seen in Fig. 2 (a), the fiber orientations gradually rotates from $+60^\circ$ at the endocardium with respect to the circumferential direction to -60° at the epicardium. On the other hand, we make use of the cubic-Hermite mesh topology to define the fibers in the atria (Fig. 2(b)) which are transmurally constant.

2.3. Simulation of full cardiac cycle

A complete cardiac cycle was simulated by coupling the finite element mesh of a two-chamber human cardiac model with a lumped-parameter closed-loop circulation model using the methods described in Kerckhoffs et al. (2007). The pulmonary and systemic circulations are each modeled as two lumped Windkessel compartments in series. The model couples both the 3D ventricular finite element model and the lumped-parameter model with the assumption of constant blood volume inside the cardiovascular system. Convergence is achieved if the difference between the calculated ventricular volumes obtained from both models at each time step lies within a tolerance range (10^{-4}).

We make use of the model developed by Holzapfel and Ogden (2009) to model the passive material properties of the cardiac tissue. The strain energy in this model is given by

$$\psi = \frac{a}{2b} e^{b(I_1-3)} + \frac{a_f}{2b_f} \left(e^{b_f(I_{4f}-1)^2} - 1 \right). \quad (1)$$

In Eq. (1), I_1 corresponds to the first invariant of the right Cauchy-Green strain tensor, I_{4f} corresponds to the components of the right Cauchy-Green strain tensor in the fiber direction. The parameter values used for the simulations in this paper are listed in Table 2.

Javani et al. (2016) studied the passive mechanical properties of a healthy ovine heart using a planar biaxial stretching system. They showed that the stress-strain response of all four different chambers' specimens is nonlinear both in fiber and cross-fiber directions. Considering the Fung strain energy function to fit the material coefficients, they obtained stiffer behavior for atria compared to the ventricles (up to $1.5\times$). In the present work, we assumed that both the left and the right atria behave stiffer than ventricles (two times stiffer) by increasing the coefficients of the corresponding strain energy function (a_f and b_f) in Eq. (1).

The active contraction model developed by Lumens et al. (2009) is used to model muscle contraction. This active contraction is defined as a function of sarcomere length (L_s) and mechanical activation (C) by

$$\sigma_{f,act} = \sigma_{act} C (L_{sc} - L_{sc0}) \frac{L_s - L_{sc}}{L_{se,iso}}. \quad (2)$$

The details of the active contraction stress can be found in Lumens et al. (2009) and it is defined as a 1-D model. Assuming no shear stresses during contraction, this mid-wall tension is converted to

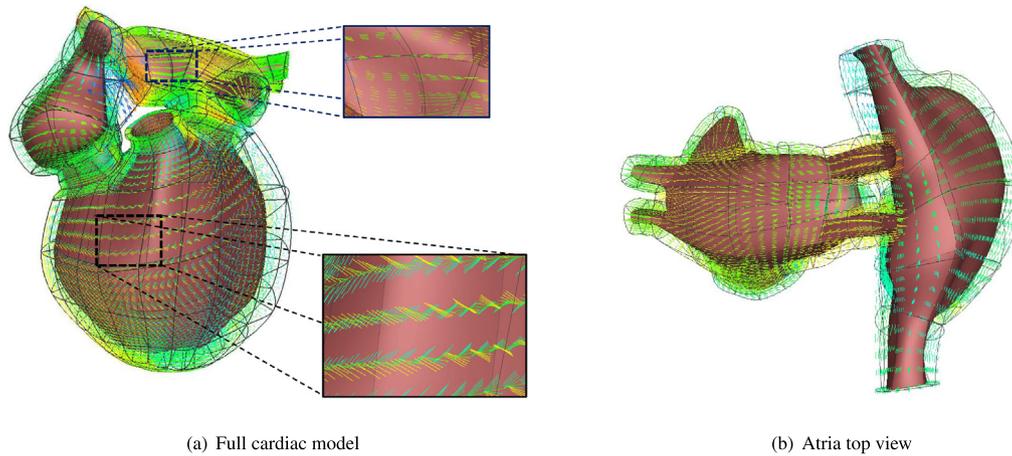


Fig. 2. Fiber orientation from $+60^\circ$ (at endocardium) with respect to the circumferential direction to -60° (at epicardium) at ventricular region; the Atrial fiber angle is kept at zero everywhere, following the cubic-Hermite mesh topology.

Table 2

Passive parameters of the Ogden-Holzapfel models.

	a (kPa)	b	a_f (kPa)	b_f
Present work	0.684 (LV, RV) 1.368 (LA, RA)	9.726	0.51 (LV, RV) 1.02 (LA, RA)	15.779
Krishnamurthy et al. (2013b)	0.684	9.726	0.51	15.779
Holzapfel and Ogden (2009)	2.28	9.726	1.685	15.779

a 3 dimensional transversely isotropic active stress with the transverse component being 30% of the fiber direction active stress (Guccione et al., 1991) as follows

$$\mathbf{T}_{active} = \begin{bmatrix} \sigma_{f,act} & 0 & 0 \\ 0 & 0.3\sigma_{f,act} & 0 \\ 0 & 0 & 0.3\sigma_{f,act} \end{bmatrix}. \quad (3)$$

The T_{active} components in Eq. (3) is added to the passive stress matrix calculated from the strain energy. The combined stress equation is then used to solve for the deformed geometry that will be in equilibrium with the externally applied pressure boundary conditions on the cardiac walls.

In this work, we consider the time-dependent variation of the atrial pressures, deformation, and contraction. The electrical signal generated in the sinoatrial node travels through the atria causing the atrial muscles contract first while the ventricles contract 120 ms later in a normal human heart. In the present work, we applied the timing difference in atrial and ventricular contraction by modifying the corresponding active tension's starting time (Eq. (2)). This modification in active tension for ventricular and atrial chambers let the atria contract 120 ms in advance of ventricular contraction. All regions of the ventricles were contracted simultaneously.

The circulation model is coupled to the two chambers of the four-chamber model, since this coupling has to conserve the volume of the ventricles due to the presence of valves. We ran the simulation for seven full beat cycles, which was enough to achieve a steady state for the cardiac simulations. The circulation model outputs the atrial pressure as well as the ventricular pressure values, which are used as pressure boundary conditions to achieve the deformation of the four-chamber cardiac model for the full beat cycle. Owing to the complex geometry of a four-chamber cardiac model, the necessary and proper boundary conditions need to be carefully applied to replicate the deformation of the heart. In our model, both Mitral and Tricuspid valves (displayed in orange color in Fig. 3(a)) were fixed along the x-axis (vertical direction) while

they can move freely in other directions (y-axis and z-axis). Moreover, the first and second-cross derivatives with respect to y and z directions were set to zero to prevent any non-planar deformation near the valves. The Pulmonary artery and Aorta (displayed in blue color in Fig. 3(a)) are only fixed in y and z directions while they can move freely along the x-axis (vertical direction). The first and second derivatives with respect to y and z directions are set to zero to avoid any non-planar deformations. The venae cavae and pulmonary veins are fixed in three directions without applying any first or second order derivatives (green colored nodes). The Neumann pressure boundary conditions were applied on the inner surfaces of all the four chambers as shown in Fig. 3(b). These boundary conditions prevent any rigid body motion of the heart while not constraining any other specific region, making them equivalent to the *in vivo* rigid body constraints.

2.4. Simulation with myocardial infarction

We studied a heart with an infarcted region (Fig. 5(a)) to investigate the acute effects of MI on the ventricular efficiency and the corresponding acute P-V loop. The volumetric ratio of the infarcted region compared to the left ventricular wall volume is about 15%. MI leads to the stiffening (chronic effects) of the myocardium in the infarcted region along with a reduction in its contractility (acute effects). We modeled the acute tissue damage by keeping the muscle stiffness the same as the healthy heart, while reducing the contractility σ_{act} in Eq. (2) down to ten times less than a healthy heart (Genet et al., 2015).

3. Results

3.1. Validation of cardiac motion

The validity of the proposed rigid body constraints to replicate the correct normal cardiac motion can be examined by comparing the normalized apex-base and apex-atrium distances (with respect

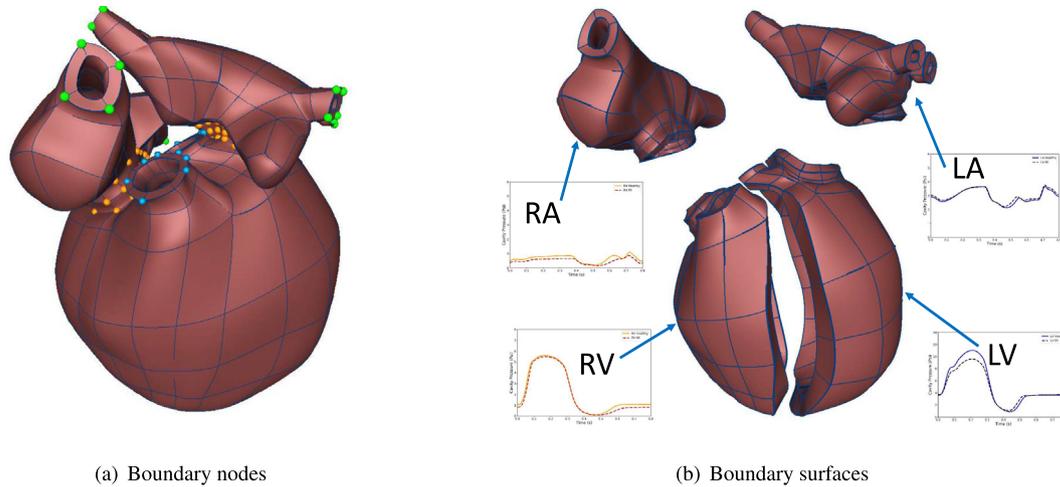


Fig. 3. Boundary conditions.

to their corresponding maximum values) obtained from our simulation with open-source MRI data provided by Kaggle Second Annual Data Science Bowl (2015). The comparison between the model and the clinical data over the complete cardiac cycle (Fig. 4(c)) shows that simulation results closely match the clinical data (Error $RMS_{apex-base} = 1.5\%$, $RMS_{atrium-apex} = 1.7\%$).

Moreover, Kou et al. (2014) performed a comprehensive echocardiographic examination of 734 healthy volunteers to provide normal reference ranges for cardiac chambers size during a full-beat cycle. The comparison between the ratio of the right ventricle basal linear dimension (RV_b) at the end-diastole and end-systole obtained from the present work (1.62) is within the range of values reported by Kou et al. (2014) (1.45 ± 0.13) (Table E.4 and Fig. E.9).

3.2. Comparison of PV-loops and cardiac motion

The comparison between the PV-loop of a healthy heart and the heart with MI shows that there is a significant reduction in the acute left ventricular peak pressure ($\approx 13\%$, Fig. 6(a) and (e)) while

there is not that much change in the right ventricular peak pressure ($< 2\%$). This is because the MI is confined to the left ventricular walls. Both the left and the right ventricular volumes are larger at end-systole for a heart with MI compared to the healthy heart, since an acute myocardial infarction reduces local contractility (in this case, the left ventricle) (Fig. 6(b)). However, even though the myocardial infarction is localized to the left ventricular wall, the right ventricular PV-loop would still be affected due to the constant blood volume in the circulation system. In addition, as Fig. 6(c) shows, the right atrial pressure history of the heart with MI is slightly lower than the one corresponding to the healthy heart.

The decrease in pumping ability due to MI is calculated by the area enclosed inside the PV-loop (Fig. 6(e)) for both ventricular chambers. As stated earlier, the acute MI causes the loss of contractility for the left ventricle (local effects). Since we modeled only the acute effects, we did not assume any material stiffness change for the infarcted region. The right ventricle fills up to less volume at end-diastole for MI compared to the healthy heart. This can be explained by the fact that the constant blood volume inside the circulatory system necessities this loss in filling for the right ventricle.

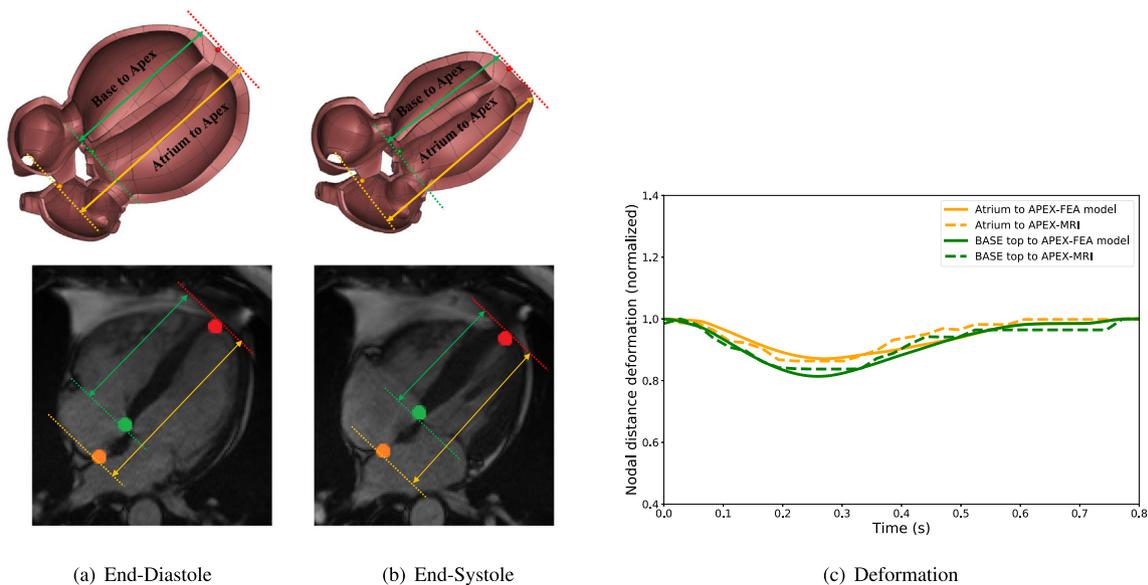


Fig. 4. Cut-section view of the cardiac model and open-source clinical data (Kaggle Second Annual Data Science Bowl, 2015) at end-diastole and end-systole states. Comparison between the normalized apex-base and atrium-apex with clinical data in a cardiac cycle, $RMS_{apex-base} = 1.5\%$, $RMS_{atrium-apex} = 1.7\%$.

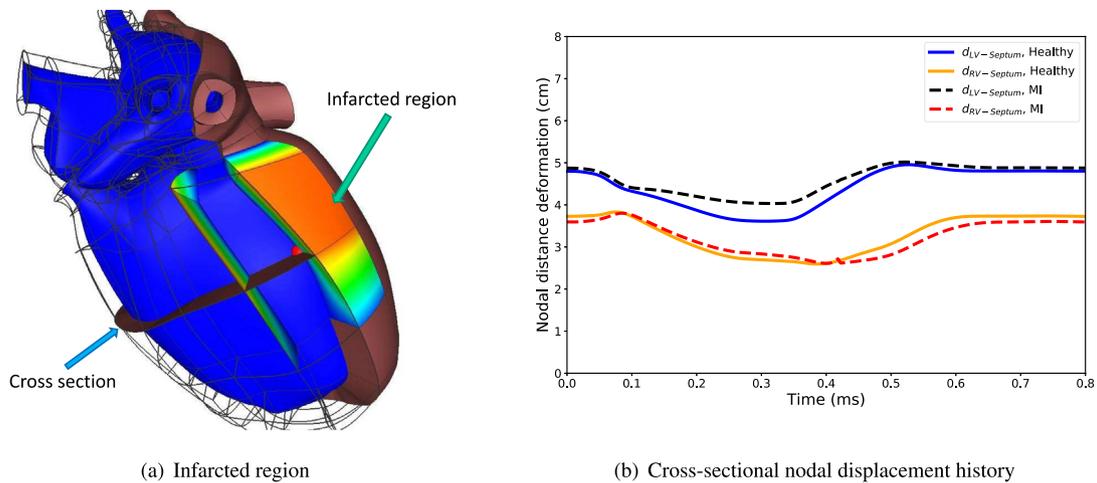


Fig. 5. Heart with myocardial infarction.

Due to the less active cardiac cells in the heart with MI, its pumping ability is reduced by 20% compared to the healthy heart (16% reduction from the left ventricle and 4% from the right ventricle, see Table 3).

The changes in the left and the right ventricles' deformation in the heart with MI and the healthy heart at different states of the cardiac cycle is shown in Fig. 7. The left column shows the cross-sectional views of a healthy heart while the right column shows the heart with MI. The nodal distance between the septum and the left and the right ventricular free wall, denoted by $d_{LV-Septum}$ and $d_{RV-Septum}$, can be used to determine the effects of MI on the local ventricular deformation. As can be seen, the LV in a heart with MI fills as much as a healthy heart (Fig. 7 (a) and (b)) at end-diastole, while at peak pressure and the end-systolic state, it shows less contraction (Fig. 7 (c) and (d)) due to the weaker contractility of the heart with MI. At lower pressure (Fig. 7 (g) and (h)), this difference is negligible compared to the other cardiac state. On the other hand, the RV in a heart with MI expands less than the healthy heart at end-diastolic states. In addition, as a consequence, the RV also loses some of its contractility due to the Frank-Starling mechanism.

Fig. 5(b) shows the history of $d_{LV-Septum}$ and $d_{RV-Septum}$ in the cardiac cycle. As can be seen the differences in $d_{LV-Septum}$ between a heart with MI and a healthy heart is more significant when the heart is contracting while the right ventricle shows greater deviation from the healthy heart during isovolumic contraction after the end-diastolic state. This shows that losing the contractility is more critical for the LV at end-systolic state while the effects are more prominent on the RV after the end-diastolic state.

4. Discussion

The heart's complex geometry requires the implementation of very fine meshes on regions of high curvature, which makes simulations numerically expensive with linear elements. For instance, Augustin et al. (2016) presented an accurate high-resolution model of the human heart electromechanics by using up to 184.6 million tetrahedral elements to solve the nonlinear governing equations. However, the use of cubic-Hermite elements in our study helps us to use fewer number of elements to capture the complex geometry. In addition, the mesh element sizes that we have employed for our four-chamber model are refined enough to obtain converged mesh displacements and chamber volumes (see Appendix F for mesh size comparison and convergence of cubic-Hermite

elements for biomechanics). We had previously reported that cubic-Hermite meshes require fewer Newton-Raphson iterations to converge even in the presence of extraordinary nodes (Krishnamurthy et al., 2015). Recent comprehensive work done by Vincent et al. (2015) compares the convergence behavior of different interpolation methods for finite element simulations on a cardiac monodomain equations for electrophysiology. These convergence analysis results show that cubic-Hermite meshes can accurately capture the biomechanics of a cardiac geometry with fewer elements.

Fritz et al. (2014) built a four-chamber cardiac model from MRI data of a healthy middle age volunteer to study the interaction between the ventricles, the atria, and the pericardium in a full-beat cycle. By developing a contact handling algorithm, they were able to solve the contact between the epicardium and the pericardium. The apex, the openings of the pulmonary vein, and both the inferior and the superior venae cavae, were fixed in their model as well as the outer surface of the mesh of the surrounding tissue. They found out that, after including the pericardium, the contour of the outer surface of the heart of varied only minimally, although the ventricles, as well as the atria, were significantly deformed. In this study, we have not included the effect of pericardium directly. One possible way this can be included in our model is to apply direct pressure to the outer faces of the elements that can simulate the effect of the pericardium. However, even without this effect included, we found that the apex-base displacement in our model matches commonly observed values reported in the literature. In addition, we post processed our deformation results to fix the apex instead of the valve plane (please see included full beat video, Online supplement Appendix D) The resulting deformations match observed cardiac deformations in the chest cavity.

There have been several related work on computational cardiac modeling. Please see the online supplement for a detailed discussion of these additional related works (Arsigny et al., 2005; Bernus et al., 2002; Bourdin et al., 2007; Bradley et al., 1997; Catmull and Clark, 1978; Culver, 1966; DeRose, 1990; Doo and Sabin, 1978; Du and Schmitt, 1990; Farin, 1982; Farin, 1986; Fillard et al., 2006; Freeman et al., 1985; Gasser and Forsell, 2011; Holmes et al., 2000; Hughes et al., 2005; Land et al., 2017; Li et al., 2005; Liu and Hoschek, 1989; McLeod, 1977; Nielsen et al., 1991; Pathmanathan et al., 2012; Pennec et al., 2006; Perk et al., 2012; Petitjean and Dacher, 2011; Pfaller et al., 2019; Remme et al., 2004; Rijcken et al., 1999; Ringenberget al., 2014; Robb and Robb, 1942; Rogers and McCulloch, 1994; Sáez and

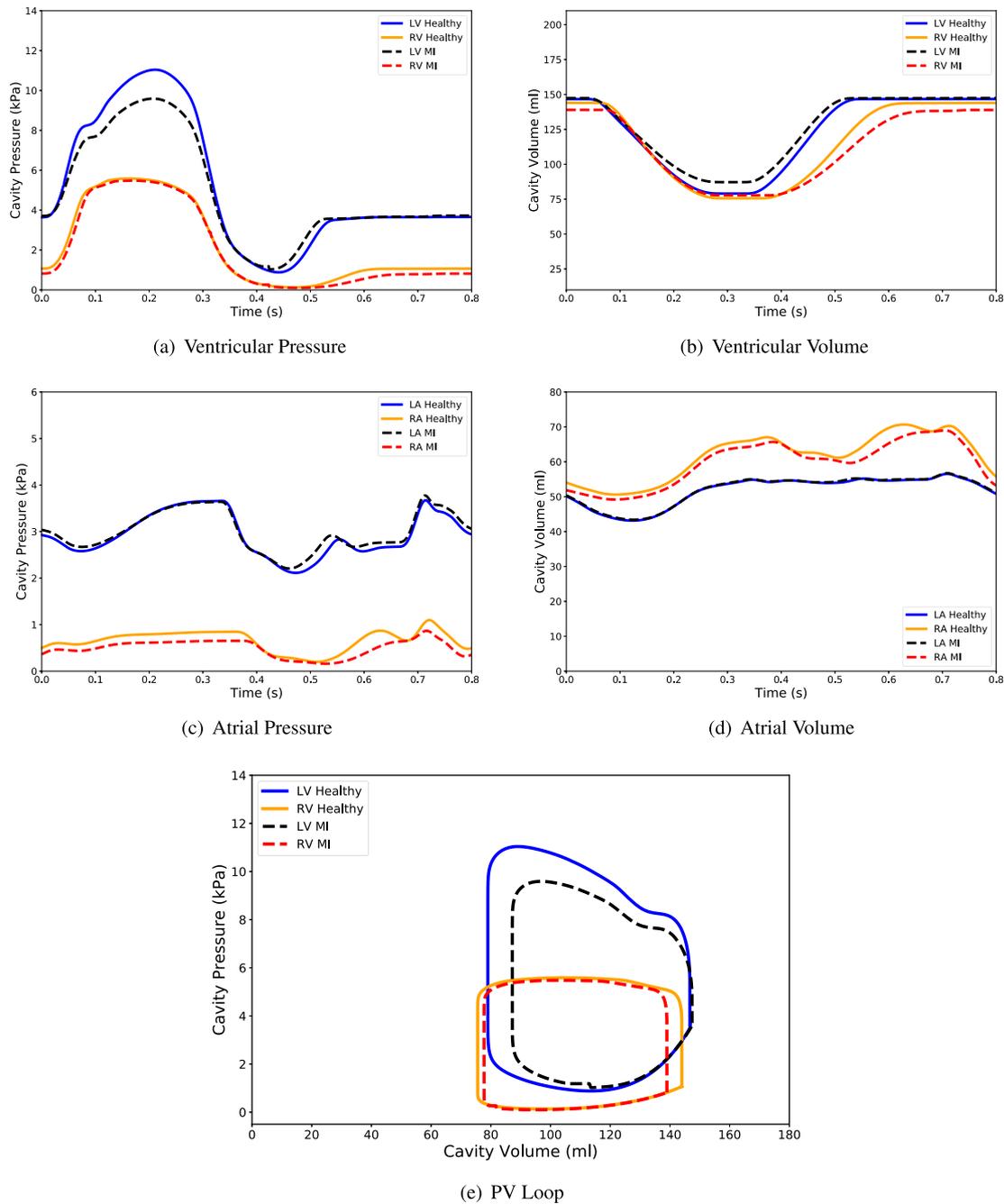


Fig. 6. Pressure and volume time-course output from the full beat simulation of the four-chamber heart model. (e) shows the simulated pressure-volume loops for the left and the right ventricles of a healthy heart and a heart with MI.

Table 3
Characteristics of the healthy heart and the heart with MI.

	LV Work	RV Work	Peak Pressure	Lowest Pressure
Healthy Heart	557 (kPa ml)	345 (kPa ml)	11.04 (kPa)	5.58 (kPa)
Heart with MI	414 (kPa ml)	309 (kPa ml)	9.59 (kPa)	5.47 (kPa)

Kuhl, 2016; Shioura et al., 2007; Smith et al., 2008; Sommer et al., 2015; Taber, 1995; Toussaint et al., 2013; Vetter and McCulloch, 1998; Zhukov and Barr, 2003)).

We have presented a four-chamber cardiac model utilizing cubic-Hermite elements and simulated a full cardiac cycle by coupling the 3D finite element model with a lumped-parameter circu-

lation model. The G^1 continuity of the finite-element fields in the neighborhood of extraordinary nodes was maintained using an ensemble coordinate system with a linear global-to-local transformation. The myocardial fiber orientations were interpolated within the mesh using the Log-Euclidean transformation to overcome the singularity associated with interpolation of orthogonal matrices.

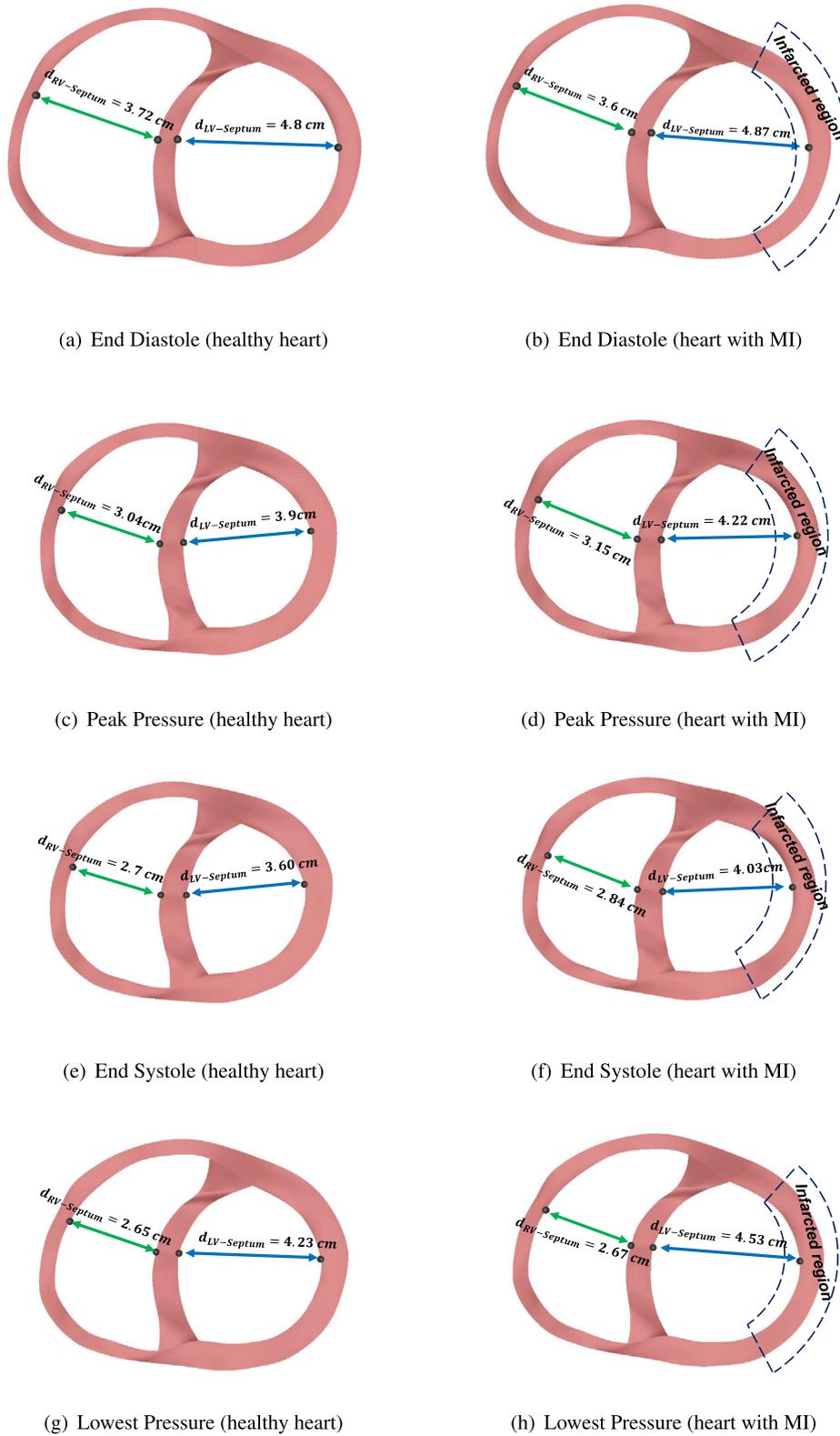


Fig. 7. Cross-section views of the infarcted region at different cardiac cycle for healthy heart and the heart with MI.

Physiologically equivalent rigid body constraints were applied to the nodes along the valve plane. We simulated a complete cardiac cycle of a healthy heart using this four-chamber model. Accurately modeling the geometric structures of the heart allows the applica-

tion of practical and physiologically equivalent rigid body constraints. These, in turn, allows the model to replicate the deformations of the different regions of the heart. The resulting deformations were validated using open-source cardiac motion

data from the literature. Our four-chamber model has the capability to match patient-specific cardiac deformations, thereby improving the state-of-the-art of patient specific cardiac modeling.

Declaration of Competing Interest

The authors confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial interests for this work that could have influenced its outcome.

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Supplementary material

Supplementary data associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.jbiomech.2019.05.019>.

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