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Model of Left Ventricular Contraction: Validation Criteria and Boundary Conditions

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Abstract

Computational models of cardiac contraction can provide critical insight into cardiac function and dysfunction. A necessary step before employing these computational models is their validation. Here we propose a series of validation criteria based on left ventricular (LV) global (ejection fraction and twist) and local (strains in a cylindrical coordinate system, aggregate cardiomyocyte shortening, and low myocardial compressibility) MRI measures to characterize LV motion and deformation during contraction. These validation criteria are used to evaluate an LV finite element model built from subject-specific anatomy and aggregate cardiomyocyte orientations reconstructed from diffusion tensor MRI. We emphasize the key role of the simulation boundary conditions in approaching the physiologically correct motion and strains during contraction. We conclude by comparing the global and local validation criteria measures obtained using two different boundary conditions: the first constraining the LV base and the second taking into account the presence of the pericardium, which leads to greatly improved motion and deformation.

Keywords

Cardiac contraction; Validation criteria; Boundary conditions; MRI

1 Introduction

Heart Failure (HF) remains a widespread health problem worldwide. In order to improve our understanding of cardiac contraction in health and disease, on-going research seeks to develop electromechanical computational models based on medical imaging data.

Computational models can help uncover the mechanisms underlying HF and, based on this

improved mechanistic understanding, more effective therapies and treatment plans can be designed and proposed (see, e.g., [1, 4]). Progress toward designing new therapies for patients with HF, however, is hindered by an incomplete understanding of the normal functioning of the heart in healthy subjects. Current models of cardiac mechanics are often based on idealized assumptions (e.g., idealized ellipsoidal geometries, rule-based aggregate cardiomyocyte –“myofiber”– orientations, over-simplified boundary conditions) that limit their applicability in patient-specific simulations and/or fail to reproduce key characteristics of cardiac contraction. Recent studies [2, 11, 12] have shown the importance of including a pericardial boundary condition (BC) in reproducing realistic left ventricle (LV) motion. In this work we construct a series of boundary conditions, including a pericardial boundary condition, to replicate the *in vivo* constraints and motion [12].

Our objectives were: (1) to construct a subject-specific LV model that integrates experimental MRI data for cardiac anatomy and microstructure without using rule-based approaches; (2) to establish a set of criteria based on cardiac MRI data to validate the computed cardiac motion; and (3) to vet the simulation results obtained using the subject-specific LV model and compare against the proposed validation criteria.

2 Validation Criteria

LV systolic motion is characterized by global and local measures that a model of cardiac contraction should reproduce before it can be used to evaluate pathological conditions or plan therapeutic interventions.

2.1 Global Measures

Ejection Fraction (EF) and twist angle are global measures of cardiac function that serve as clinical markers to detect the onset and monitor the progression of cardiac diseases.

- EF: The average left ventricular EF is $\approx 56.6\% \pm 4.6$ [18] in healthy human subjects and an EF below 50% is considered a symptom of heart failure with reduced ejection fraction (HFrEF) [8]. However, normal healthy subject EF values may vary in other species.
- Twist angle: Peak LV twist equal to $11.5^\circ \pm 3.3^\circ$ has been reported in [14] for healthy human subjects. Peak twist is measured as the difference in rotation at the LV base ($-3.9^\circ \pm 1.3^\circ$) and LV apex ($7.5^\circ \pm 3.6^\circ$).

2.2 Local Measures

Strain measures are clinical biomarkers of regional cardiac function. Strains are usually reported along the longitudinal, circumferential, and radial directions, although this reference system depends on an arbitrary geometrical definition and cannot be uniquely defined. As local measures we use both the widely reported strains in a cylindrical coordinate system and strain in the direction of aggregate cardiomyocytes, which is directly related to the average cardiomyocyte shortening driving cardiac contraction. The direction of aggregate cardiomyocytes does not depend on an arbitrary geometric definition but directly

reflects the microstructure of the myocardium and can be measured using diffusion tensor imaging (DTI).

Average Green-Lagrange strain values at peak systole for the mid-ventricular LV are:

- Longitudinal strain E_{ll} : -0.16 ± 0.02 [20], -0.15 ± 0.02 [9]. Longitudinal strain corresponds to the LV base-to-apex shortening observed during contraction.
- Circumferential strain E_{cc} : -0.17 ± 0.02 [20], -0.19 ± 0.02 [9], with a gradient across the myocardial wall ($E_{cc} = -0.16 \pm 0.02$ at the epicardium and $E_{cc} = -0.20 \pm 0.02$ at the endocardium [20]).
- Radial strain E_{rr} : a larger range is present in the literature for E_{rr} with respect to other strain measures. Zhong et al. [20] report average peak systolic mid-wall $E_{rr} = 0.33 \pm 0.10$ while Moore *et al.* [9] report $E_{rr} = 0.42 \pm 0.11$. E_{rr} also exhibits a transmural gradient: $E_{rr} = 0.29 \pm 0.11$ at the epicardial wall and $E_{rr} = 0.38 \pm 0.10$ at the endocardium [20]. Radial strains correspond to the transmural wall thickening observed during contraction.
- Aggregate cardiomyocyte strain E_{ff} : it ranges from -0.13 [10] and -0.12 ± 0.01 [17] to -0.18 [18].
- Incompressibility: *in vivo* MRI-based study [15] reported 1–2% myocardial volume change during contraction. The low compressibility of the myocardium has also been confirmed by *ex vivo* tissue studies [5, 19] that reported a volume change of approximately 2–4% during contraction. This limited compressibility is attributed to blood outflow during contraction.

3 Methods

The finite element model is based on: (1) MRI-based subject-specific LV anatomy and microstructure acquired in a healthy swine; (2) a sliding boundary condition to model the interaction between visceral (epicardium) and the deformable parietal pericardium; (3) a boundary condition to model the basal surface constraint; (4) an auxiliary electrophysiology model to compute the activation times throughout the LV [6]; and (5) active and passive material laws to model the myocardial mechanical behavior during filling and ventricular systole adapted from [13]. Here, the force velocity curve in [13] governing cardiac contraction is calibrated to achieve $EF \approx 50\%$.

In the following sections, we describe the construction of the finite element model, the corresponding boundary conditions, and the calculation of quantitative measures of cardiac function at peak systole to evaluate the fulfillment of the listed validation criteria.

3.1 Subject-Specific LV Anatomy and Microstructure

The anatomy and microstructure (cardiomyocyte aggregate orientations) data of the FE model were computed from DTI of an *ex vivo* swine heart. The animal experiments were conducted in accordance to research protocol # 2015–124 approved by the UCLA Chancellor's Animal Research Committee. After euthanasia, the heart was extracted and the four chambers were filled with a silicone rubber compound to approximate the heart

configuration corresponding to the lowest intra-ventricular pressure. The heart was then submersed in perfluoropolyether solution with no MR signal and scanned overnight for eight hours (readout-segmented, diffusion weighted spin echo sequence, 30 directions, b-value = 1000 s/mm^2 , Echo Time/Repetition Time = 62 ms/18100 ms, 5 signal averages, and a spatial resolution of $1.0 \times 1.0 \times 1.0 \text{ mm}^3$).

The DTI data acquired *ex vivo* was segmented to determine the epicardial and endocardial contours that were subsequently edited to smooth surfaces (3-matic, Materialise). The myocardial volume enclosed by the endocardial and epicardial surfaces was meshed with 6109 quadratic tetrahedral elements (Fig. 1A-B) (average element edge length = 5.3 mm [95% CI: 4.8 mm, 6.5 mm], average element Jacobian = 0.97 [95% CI: 0.96, 1.0]). Diffusion tensors were reconstructed at each DTI voxel and interpolated at each mesh quadrature point using an in house Matlab code and linear tensor invariant interpolation [3]. Aggregate cardiomyocytes orientations were computed as the primary eigenvectors of the diffusion tensor at each quadrature point (Fig. 1C).

The pericardial surface was constructed by projecting outward the LV epicardial surface by a distance δ in the normal direction (Fig. 1D). The pericardial surface was then meshed using linear triangular elements.

Since shape and volume are important contributors to LV function, we verify that our *ex vivo* based geometry represents the *in vivo* LV reference configuration at the lowest intraventricular pressure, which was measured during MR exams using a fiber optic pressure transducer. The *ex vivo* myocardium was within 2.4% of the *in vivo* tissue volume measured from CINE images at diastasis, end diastole, and peak systole. The *ex vivo* LV cavity volume was within 4.2% of the *in vivo* cavity volume in the reference configuration. The computed Dice Similarity Coefficient between the *ex vivo* and *in vivo* configurations was 0.80, indicating that the differences between the two configurations are similar to differences due to intra-observer segmentation [21].

3.2 Boundary Condition: Epicardial and Basal Surfaces

The heart is connected to the great vessels and is contained in the parietal pericardium (Fig. 2) wherein it contracts and twists with minimal resistance. These boundary conditions are modeled by including: (1) a flexible surface that represents the pericardium and exerts a reaction force only in the direction normal to itself; and (2) a constraint to limit the warping and out of plane rotation of the LV basal surface due to the presence of the valves' structure and great vessels connected to the heart.

Epicardial Surface.—The pericardium is modeled as a flexible elastic membrane with a bending and in-plane stretching energies. For simplicity, the stretching and bending energies are modeled using a network of springs between the element nodes and the element normals. Although approximate, this simple approach to model elastic shells has been used in several studies in large deformations, e.g., [7]. The total pericardium elastic energy is obtained by summing the element contributions W_e^s (in-plane stretching energy) and W_e^b (bending

energy). $W_e^s = \frac{1}{2}k_p \sum_{i=1}^3 (l_e^i - L_e^i)^2$, where k_p is the spring elastic constant, $i = 1 \dots 3$ refers to the edges of a linear triangular element e , l_e^i is the current length of edge i , and L_e^i is corresponding length in the reference configuration. $W_e^b = \frac{1}{2}k_b \sum_{i=1}^3 (\|\mathbf{n}_e - \mathbf{n}_i\| - \bar{\theta}_{ei})^2$, where k_b is the angular spring elastic constant, $i = 1 \dots 3$ refers to the elements sharing an edge with element e , $\mathbf{n}_e$ and $\mathbf{n}_i$ are the unit normals to element e and i , and $\bar{\theta}_{ei}$ is the angle between $\mathbf{n}_e$ and $\mathbf{n}_i$ in the reference configuration. Consistent with the small angle approximation, $\bar{\theta}_{ei} \approx \|\bar{\mathbf{n}}_e - \bar{\mathbf{n}}_i\|$, where $\bar{\mathbf{n}}_e$ and $\bar{\mathbf{n}}_i$ are the unit normal in the reference configuration.

In order to minimize any artefactual constraints on cardiac twist and longitudinal motion, the pericardium only exerts forces in the direction normal to its surface according to the following interaction energy W^{int} :

$$W^{int} = \frac{1}{2}k_{int} \sum_{a=1}^{N^{epi}} \frac{1}{N^{search}} \sum_{b=1}^{N^{search}} ((\mathbf{x}_b - \mathbf{x}_a) \cdot \mathbf{n}_b - \delta)^4, \quad (1)$$

where k_{int} scales the interaction forces between the parietal pericardium and the LV, a and b represent a node on the epicardium and parietal pericardium, respectively, N^{search} is the number of nodes within a search R_{search}^{epi} from node a , $\mathbf{x}_a$ and $\mathbf{x}_b$ are the current nodal positions of nodes a and b , and $\mathbf{n}_b$ is the unit normal to the parietal pericardium at node b (Fig. 2). In our simulations we used: $\delta = 0.5$ cm and $R_{search}^{epi} = 1.5$ cm, but limit N^{search} to three.

The mesh of the parietal pericardium was extended above the LV basal plane (see Fig. 2) and anchored by constraining the nodes in its most basal region. Since the constrained nodes are above the LV basal plane, this boundary condition does not limit the in-plane and longitudinal motion of the LV. Similarly, the nodes in the apical region of the parietal pericardium mesh were constrained in order to prevent motion of the apex in the short-axis plane without affecting physiological LV twist.

Basal Surface.—The LV basal surface out of plane rotation and warping is constrained by the valves' structure and the great vessels connected to the heart. This constraint is represented by the following energy that penalizes deviation from the basal surface reference configuration while it allows its free translation along the longitudinal axis.

$$W^{base-n} = \frac{1}{2}k_{base-n} \int_{\Gamma_{base}} \|\mathbf{n} - \bar{\mathbf{n}}\|^2 d\Gamma, \quad (2)$$

where k_{base-n} is the bending stiffness of the LV basal surface Γ_{base} due to the valves' structure and great vessels while $\bar{n}$ and $\mathbf{n}$ are the local unit normals on the basal surface in the reference and current configurations, respectively. This energy is adapted from [16].

The great vessels and the valves' structure also limit the rigid rotation of the LV around its longitudinal axis. In order to incorporate this effect, we apply torsional springs at every node on the LV base by including the following energy:

$$W^{base-t} = \frac{1}{2} k_{base-t} \sum_{a=1}^{N^{base}} [(\mathbf{x}_a - \mathbf{X}_a) \cdot \mathbf{c}]^2, \quad (3)$$

where k_{base-t} is the torsional stiffness, N^{base} is the total number of nodes a on the basal surface, $\mathbf{X}_a$ and $\mathbf{x}_a$ are the reference and current positions of basal node a , respectively, and $\mathbf{c}$ is the in-plane (perpendicular to the longitudinal axis) circumferential unit vector.

3.3 Computing Measures of Cardiac Contraction

Based on the LV configuration at peak systole, the following output measures were computed to be compared with the reference values in the list of validation criteria:

- **LV Ejection Fraction.** EF is the ratio between the stroke volume (SV) during systole and the end diastolic cavity volume (EDV). SV is equal to the difference between EDV and ESV, where ESV is the end systolic cavity volume. All cavity volumes are computed using the divergence theorem.
- **LV Twist Angle.** Since the LV longitudinal axis is aligned here with the Z-axis and the chosen basal boundary condition constrains rotations at the LV base, LV twist is computed as the average rotation of an apical slice around Z.
- **Characteristic Strains.** Based on the deformation gradient tensor $\mathbf{F}$, we compute the Green-Lagrange strain tensor $\mathbf{E} = \frac{1}{2}(\mathbf{F}^T \mathbf{F} - \mathbf{I})$, where $\mathbf{I}$ is the identity tensor. By projecting $\mathbf{E}$ along different directions, we compute strains along the cylindrical axes $\mathbf{r}$, $\mathbf{c}$, $\mathbf{l}$, and aggregate cardiomyocyte direction $\mathbf{f}$. LV strain measures are then divided in epicardial, mid, and endocardial regions based on a continuous scalar field φ : $\Omega_{epi} = \{X: 0 \leq \varphi(X) < \frac{1}{3}\}$, $\Omega_{mid} = \{X: \frac{1}{3} \leq \varphi(X) \leq \frac{2}{3}\}$ and $\Omega_{endo} = \{X: \frac{2}{3} < \varphi(X) \leq 1\}$. φ is the solution of the Laplace equation solved in the LV domain Ω with boundary conditions $\varphi = 0$ on the epicardial wall and $\varphi = 1$ on the endocardial wall.
- **Incompressibility.** Tissue compressibility was evaluated based on $J = \det(\mathbf{F})$.

4 Results

During filling the pericardial boundary condition (Fig. 3) supports the apex while leaving the base free to move upward. The LV epicardial surface moves outward only slightly, which is qualitatively consistent with observed *in vivo* motion patterns. During contraction, the LV

with the pericardial boundary condition shows significant twist (12.5°), longitudinal shortening, and wall thickening (Fig. 3). The corresponding ejection fraction at peak systole is 51.2%. On the contrary, the LV with pinned base presents very limited twist (0.1°) and longitudinal shortening. The corresponding ejection fraction at peak systole is 53.1%. Strain values corresponding to wall thickening (E_{rr}) and longitudinal shortening ($E_{\theta\theta}$) are shown in Fig. 4 together with circumferential and aggregate cardiomyocytes strains. Both radial and circumferential strains present a transmural gradient that agrees with trends presented in the literature. The average Jacobian for both boundary conditions indicates very limited tissue volume change (pericardial boundary condition: 3.3%; pinned boundary condition: 1.6%).

5 Discussion

We have presented a set of validation criteria based on MRI measures that can help develop and validate cardiac models of LV contraction. The simulation using the pericardial boundary condition shows far more physiologically accurate cardiac motion and deformation when compared to the simulation using the pinned based boundary condition. This is despite the fact that both simulations lead to an EF close to the *in vivo* value of 48.7%. Therefore, EF alone is not sufficient to evaluate a model. The need for a correct pericardial boundary condition agrees with other studies, e.g., [2, 11]. The pericardial boundary condition allows eliminating LV rigid body motions without imposing unphysiological constraints on cardiac motion. This is possible also because the pericardial surface is not rigid but flexible, allowing the heart (which does not have an axially-symmetric geometry) to better slide while twisting during contraction. A flexible pericardial boundary condition better represents the *in vivo* conditions surrounding the heart.

The model presented here was built using subject-specific data and contained the essential components to meet the presented validation criteria. However several improvements are possible, including a direct coupling with the electrophysiology model, the inclusion of the right ventricle, and possibly the atria and great vessels to anchor the heart. Furthermore, the current pericardial boundary condition does not take into account the stiffness of the surrounding organs, such as the lungs and the liver. Here, the stiffness of the pericardial boundary condition was calibrated to minimally constrain the heart motion while maintaining convergence of the finite element model.

The presented list of validation criteria is not meant to be exhaustive and several other criteria may be considered depending also on the simulation goals. For example, additional validation criteria may focus on the passive filling, active relaxation phase, and/or may include measures of aggregate cardiomyocytes kinematics. In addition, the validation criteria listed here have been derived from literature mostly focusing on human data. Inter-species variability should also be taken into account and species-specific reference values for the listed validation criteria should be used as they become available.

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Fig. 1. Model of LV Anatomy and Microstructure.

(A) Segmentation of LV anatomy from *ex vivo* DTI data. (B) Generation of smooth LV surface from segmented contours and finite element discretization of LV volume. Mesh is constructed using quadratic tetrahedral elements. (C) Incorporation of aggregate cardiomyocyte orientations into LV volume mesh. Aggregate cardiomyocyte orientations are interpolated from DTI voxels to all mesh quadrature points using linear tensor invariant interpolation. Colorbar represents the aggregate cardiomyocytes elevation angle. (D) Construction of the pericardial surface boundary condition (red) enclosing the LV myocardium (yellow). (Color figure online)

Fig. 2.

(A) Parietal pericardium during post euthanasia heart extraction. (B) Long axis view of finite element mesh of the myocardium (yellow) with surrounding pericardial mesh (red). (C) Zoomed in view of epicardial and pericardial surfaces with components used to model the pericardium boundary condition (see Eq. 1). (Color figure online)

Fig. 3. LV motion from diastasis (A), through late filling (B), to peak systole (C). Section of the LV FE model with pericardial boundary condition (top) and LV outline superimposed to long-axis MR images (bottom). (D) Comparison of LV cross-sectional deformation obtained at peak systole with pinned and pericardial boundary conditions.

Fig. 4.
Peak systolic strains obtained with pericardial and pinned base boundary conditions.