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Image-Based Patient-Specific Ventricle Models with Fluid-Structure Interaction for Cardiac Function Assessment and Surgical Design Optimization

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Abstract

Recent advances in medical imaging technology and computational modeling techniques are making it possible that patient-specific computational ventricle models be constructed and used to test surgical hypotheses and replace empirical and often risky clinical experimentation to examine the efficiency and suitability of various reconstructive procedures in diseased hearts. In this paper, we provide a brief review on recent development in ventricle modeling and its potential application in surgical planning and management of tetralogy of Fallot (ToF) patients. Aspects of data acquisition, model selection and construction, tissue material properties, ventricle layer structure and tissue fiber orientations, pressure condition, model validation and virtual surgery procedures (changing patient-specific ventricle data and perform computer simulation) were reviewed. Results from a case study using patient-specific cardiac magnetic resonance (CMR) imaging and right/left ventricle and patch (RV/LV/Patch) combination model with fluid-structure interactions (FSI) were reported. The models were used to evaluate and optimize human pulmonary valve replacement/insertion (PVR) surgical procedure and patch design and test a surgical hypothesis that PVR with small patch and aggressive scar tissue trimming in PVR surgery may lead to improved recovery of RV function and reduced stress/strain conditions in the patch area.

Keywords

Right ventricle; congenital heart disease; tetralogy of Fallot; heart model; fluid-structural interaction

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1. Introduction

Patients with repaired Tetralogy of Fallot (ToF), a congenital heart defect which includes a ventricular septal defect and severe right ventricular outflow obstruction, account for the majority of cases with late onset RV failure. With recent advances in medical image technology and computational modeling techniques [1–10], attempt of using computational “virtual surgery” has been made to replace empirical and often risky clinical experimentation to examine the efficiency and suitability of various reconstructive procedures in diseased hearts [7–9]. Considerable information about myocardium tissue material properties, ventricle multi-layer structure, myofiber architecture, active stress and sarcomere length has been obtained from animal models and in vitro/ex vivo experiments [11]. However, much of the information needed for patient-specific modeling would not be readily available in clinical practices. How to construct patient-specific ventricle models based on limited information and make reliable assessment of ventricle function is a great challenge to the cardiovascular research community.

Several reviews on surgical management and cardiac magnetic resonance (CMR) imaging have been published [1,12–14]. As recommended by the National Heart, Lung and Blood Institute (NHLBI) Working Group on Future Direction in Cardiac Surgery [15], multi-disciplinary efforts are needed to integrate innovative surgical procedures, imaging technology, and patient-specific computational modeling for improved outcomes of surgical interventions for cardiovascular diseases such as ventricular restoration, valve replacement, coronary bypass surgery, etc. In this paper, we will provide a short review of recent advances in mechanical ventricle modeling as well as our effort in constructing patient-specific computational ventricle models based on in vivo cardiac magnetic resonance (CMR) imaging for surgical planning and optimization. Data required for in vivo image-based patient-specific ventricle modeling, important modeling considerations and critical controlling factors, list of surgical, morphological, and mechanical parameters which may affect assessment accuracy and surgical outcome will be discussed.

2. Background and literature review

Ventricle modeling is challenging because: a) ventricle geometries are complex; b) heart motion is complex and involves active contraction, large deformation, and strong fluid-structure interactions; c) myocardium tissue mechanical properties are complex; d) ventricle wall has complex multi-layer structure and myofiber architecture; e) valve mechanics is complex; f) data acquisition under in vivo condition is often difficult or near impossible. Diagram 1 lists three essential areas of data needed for ventricle model construction: morphology, material properties, and mechanical flow, pressure and stress conditions. Development of computational ventricle modeling and experimental research can be divided into several stages: a) basic scientific research using idealized geometries and in vitro experimental testing; b) more realistic modeling including ventricle motion, passive and active contraction and fiber orientation; c) patient-specific modeling with combination of in vivo data and data from literature for surgical planning and optimization. While research at each stage has its specific goals, our effort has been focused on clinical and surgical applications [7–9]. Our surgical hypothesis, model design and investigation process will be reviewed in this paper.

Early 3D models for blood flow in the heart include Peskin’s model which introduced fiber-based LV model and the celebrated immersed-boundary method to study blood flow features in an idealized geometry with fluid-structure interactions [16–18]. A large amount of effort has been devoted to quantifying heart tissue mechanical properties and fiber orientations mostly using animal models [19–25]. More recent efforts include introduction of Magnetic

Resonance Imaging (MRI)-based fluid-only or structure-only 3D models to investigate flow and stress/strain behaviors in the whole ventricle (either RV or LV) [7–10,26–30]. Wenk et al. created a finite element sheep model of the left ventricle (LV) with mitral apparatus, and chordae tendineae from magnetic resonance images from a sheep that developed moderate mitral regurgitation after posterobasal myocardial infarction and their results indicated that the LV model including mitral valve using noninvasive techniques may be useful for clinical investigations [26]. Long et al. constructed patient-specific flow-only LV models for 6 healthy volunteers based on MR images. In their model, LV wall movements were specified. Detailed flow patterns were reported [27]. Stevens et al. introduced a 3D finite element solid model of the heart based on measurements of the geometry and the fiber and sheet orientations of pig hearts. The end-diastolic deformation of the model was computed using the “pole-zero” constitutive law to model the mechanics of passive myocardial tissue specimens. The sensitivities of end-diastolic fiber-sheet material strains and heart shape to changes in the material parameters were investigated [31–32]. McCulloch et al. performed extensive research for 3D ventricular geometry and myofiber architecture of the rabbit heart. Their work included experimental and modeling studies of 3D cardiac mechanics and electrophysiology [22,29–30,33–34]. In a series of papers, Guccione et al. introduced anisotropic passive and active ventricle models where an additional tension term was added to the stress field to model active heart contractions [35–39]. The papers by Nash and Hunter [20] and Hunter et al. [40] provided comprehensive reviews for heart modeling, including tissue properties, fiber orientation, passive and active mechanical models, electromechanical models, and whole heart models. Those animal and human ventricle models provide insight for further more complete modeling and human ventricle mechanics and function investigations.

3. Ventricle modeling for surgical management of Tetralogy of Fallot (ToF)

When constructing ventricle models for surgical management of Tetralogy of Fallot (ToF) or other clinical and surgical applications, it should be understood that we are subjected to severe limitations under clinical practice, mainly limitation on data acquisition under in vivo conditions and available computing power and human effort. At the same time, the model and its predictions need to be “validated” before it can be used for surgical planning and optimization. Diagram 2 gives a flow chart of the modeling procedures. First, there should be a surgical hypothesis to be tested, or a clinical problem to be addressed. Then in vivo data as well as generic data from literature should be acquired or selected for model construction. The model should capture the essential aspects of the biological system and be able to make reliable and accurate predictions of a hypothetical surgery, but some simplifications should be allowed so that the model could be constructed and solved with available data and resources. The third key point is that well-accepted “end points” for assessment of surgical outcome should be selected. With that, we should be able to conduct computational simulations. The model must be first validated by patient data before it can be used to perform “virtual surgery”, which means new models are made with computer-altered ventricle morphology and used to predict the possible outcome if actual surgery were made following the altered morphology. This general procedure could be applied to various surgical applications. Publications using this procedure for ventricle surgical design are still rare in the current literature.

The above procedure was followed in our recent effort in testing a surgical hypothesis that an optimized RV volume reduction and scar tissue trimming in pulmonary valve insertion (PVI) and replacement may lead to improved recovery of RV function (see Fig. 1) [12]. While a clinical trial was going on testing this hypothesis surgically (NIH–HL63095 (PJdN), NIH–NHLBI 5P50HL074734 (PI-Geva; Co-Investigator – del Nido), a computational modeling approach was also adopted to test this hypothesis for surgical design optimization

by “virtual surgery” using patient-specific computational models (NIH-R01-HL 089269 (del Nido, Tang, Geva). In this approach, pre-operation patient-specific ventricle morphology and pressure data were acquired using non-invasive CMR. Anisotropic material properties and fiber orientation were determined using data in the literature and model parameters were adjusted to match CMR-measured ventricle volume data. The model included right and left ventricles, scar, patch, anisotropic material properties, fiber orientation and two-layer construction. Fluid-structure interaction was included which gave both flow and structural stress/strain distributions that could serve as basis for many further investigations. Ventricle stroke volume and ejection fraction (to be defined later) were selected as the “end points” for ventricle function and surgical outcome assessment. Virtual surgery was performed by adjusting the pre-operative morphology (equivalent to scar tissue trimming in actual surgery) to predict post-operation outcome and model predictions were verified by post-operation CMR measurements. Details of the models and methods are given in the following sections.

4. Data acquisition, Models, Solution Methods, and Virtual Surgery

Our model included right and left ventricles, patch and scar tissues, isotropic and anisotropic material properties, two-layer structure and fiber orientation, and passive and active contractions. Details of those features are presented below.

4.1 Data acquisition and 3D geometry re-construction

A recent detailed review of CMR imaging techniques for congenital heart disease was given by Brown, Powell and Geva [1]. For our study, cardiac MRI (CMR) studies were performed in a dedicated MRI suite located in the Department of Cardiology at Children's Hospital Boston to acquire patient-specific ventricle morphology, flow velocity, and flow rate for patients needing pulmonary valve replacement operations before and after scheduled surgeries and healthy volunteers. Using ECG-gated steady state free precession cine MRI, 12–14 contiguous slices were obtained in the ventricle short-axis plane covering the left and right ventricles from the plane of the AV valves through the apex. In each location, 30 phases were acquired during one cardiac cycle. Figure 2 depicts a sketch of a healthy human heart with RV, LV, a stack of RV contours, and the re-constructed RV/LV geometry obtained from a healthy volunteer. Figure 3 shows pre-operative CMR images and segmented MRI contour plots from a patient with repaired TOF. Figure 4 gives the stacked MRI contours and RV/LV inner/outer surface plots of the patient ventricle data showing patch, scar, and valve positions. The location and sizes of the patch and scar tissues were determined by Dr. del Nido with his experience and confirmed during actual surgery. 3D RV/LV geometry and computational mesh were constructed under the ADINA computing environment following the procedures in [7–9].

4.2 The fluid model

Blood flow in the right ventricle was assumed to be laminar, Newtonian, viscous and incompressible. The Navier-Stokes equations with ALE formulation were used as the governing equations. Pressure conditions were prescribed at the tricuspid (inlet) and pulmonary (outlet) valves (see Fig. 5 and [41–42]). To simplify the computational model, the cardiac cycle was split into two phases: **a)** The filling phase (diastole) when blood flows into the RV, the inlet was open and the outlet was closed; **b)** The ejection phase (systole) when blood was ejected out of the RV, the outlet was kept open and the inlet was closed. When the inlet or outlet was closed, flow velocity was set to zero and pressure was left unspecified. No-slip boundary conditions and natural force boundary conditions were specified at all interfaces to couple fluid and structure models together [43–44]. The complete fluid model was given in [7–9] and is omitted here.

4.3 The passive anisotropic solid model

While the right ventricle was our research focus and it included fluid-structure interaction, the left ventricle was included to provide structural support to the right ventricle so that no artificial boundary condition at the ventricular septum would be needed to keep the wall between RV and LV in the appropriate position and shape. Those artificial boundary conditions would be hard to set and justify. Left ventricle model was structure-only and proper LV pressure conditions were prescribed to generate CMR-measured LV deformation. The RV and LV materials were assumed to be hyperelastic, anisotropic, nearly-incompressible and homogeneous. Both isotropic and anisotropic materials were used in our models for comparison purposes. Scar tissue and patch material were assumed to be hyperelastic, isotropic, nearly-incompressible and homogeneous. The governing equations for the structure models are:

$$\rho v_{i,\alpha} = \sigma_{ij,j}, \quad i, j = 1, 2, 3; \text{sum over } j, \quad (1)$$

$$\varepsilon_{ij} = (v_{i,j} + v_{j,i} + v_{\alpha,i} v_{\alpha,j}) / 2, \quad i, j, \alpha = 1, 2, 3, \quad (2)$$

where σ is the stress tensor, ε is the strain tensor, v is solid displacement vector, $f_{\bullet,j}$ stands for derivative of f with respect to the j th variable, and ρ is material density. Equations (1)–(2) are used for RV/LV muscle, patch, and scar tissue.

The nonlinear Mooney-Rivlin model was used to describe the nonlinear anisotropic and isotropic material properties of the material with parameter values chosen to match experimental data available and adjusted to reflect stiffness variation of different materials [11,22,24]. The strain energy function for the isotropic modified Mooney-Rivlin model is given by [7–9,43,44]:

$$W = c_1(I_1 - 3) + c_2(I_2 - 3) + D_1[\exp(D_2(I_1 - 3)) - 1], \quad (3)$$

where I_1 and I_2 are the first and second strain invariants given by,

$$I_1 = \sum C_{ii}, \quad I_2 = \frac{1}{2} [I_1^2 - C_{ij}C_{ij}], \quad (4)$$

$C = [C_{ij}] = X^T X$ is the right Cauchy-Green deformation tensor, $X = [X_{ij}] = [\partial x_i / \partial a_j]$, (x_i) is current position, (a_i) is original position, c_i and D_i are material parameters chosen to match experimental measurements [11,22,24]. The strain energy function for the anisotropic modified Mooney-Rivlin model anisotropic model was obtained by adding an additional anisotropic term in Eq. (3):

$$W = c_1(I_1 - 3) + c_2(I_2 - 3) + D_1[\exp(D_2(I_1 - 3)) - 1] + 1/2(K_1/K_2) [\exp[K_2(I_4 - 1)] - 1], \quad (5)$$

where $I_4 = C_{ij}(\mathbf{n}_f)_i(\mathbf{n}_f)_j$, C_{ij} is the Cauchy-Green deformation tensor, $\mathbf{n}_f$ is the fiber direction, K_1 and K_2 are material constants [43–45]. Parameter values in (5) can be chosen to fit the following material models given in [22,43–45].

$$W = \frac{C}{2}(e^Q - 1), \quad (6)$$

$$Q = b_1 E_{ff}^2 + b_2 (E_{cc}^2 + E_{rr}^2 + E_{cr}^2 + E_{rc}^2) + b_3 (E_{fc}^2 + E_{cf}^2 + E_{fr}^2 + E_{rf}^2), \quad (7)$$

where E_{ff} is fiber strain, E_{cc} is cross-fiber in-plane strain, E_{rr} is radial strain, and E_{cr} , E_{fr} and E_{fc} are the shear components in their respective coordinate planes, C , b_1 , b_2 , and b_3 are parameters to be chosen to fit experimental data. The patient-specific stress-stretch curves derived from the modified Mooney-Rivlin models fitting CMR-measured data are given by Fig. 6. Figures 6(c)–(d) show that model-predicted RV volumes had good agreement with CMR data.

4.4 Active anisotropic material model

Modeling active heart contraction is much harder because stress in a pumping human heart cannot be measured in vivo non-invasively (at least not with available technology in current practice). Heart expansion/contraction can be considered as combination of passive elastic expansion/contraction caused by blood pressure and active contraction/relaxation caused by fiber stiffening/relaxation. In our approach, material stiffening in the fiber direction was used to model active contraction. Fluid-structure interaction plays an important role in the process. During the filling phase, RV fibers relaxation and increase of RV diastolic blood pressure lead to expansion of the ventricle. In the ejection phase, RV material stiffens and pulmonary valve opens. That leads to RV contraction and blood ejection. Since it is hard to separate and measure the passive and active stresses/strains in clinical practice, we chose to specify time-dependent material stiffness parameters to model RV tissue stiffening and active RV contraction. RV muscle fibers would contract/relax by following a time-dependent stiffening/relaxation material model. Fig. 6 (b) shows end-diastolic and end-systolic stress-stretch curves for the active anisotropic model. The time-dependent stiffness parameters were numerically determined to match CMR measured RV volume curves.

4.5 Two-layer model with fiber orientation

As patient-specific fiber orientation data is not yet available in current practice, we chose to construct a two-layer RV/LV model and set fiber orientation angles using fiber angles given in Hunter [40]. The angles can be adjusted in our model easily for every location when patient-specific fiber orientation data becomes available [46]. Figure 7 shows fiber orientations on a patient-specific RV/LV model and a segment of the two-layer model between two slices. Fiber orientation data from [46] indicated that fiber orientations in human heart follow similar patterns as shown in Fig. 7.

4.6 Solution method and virtual surgery procedures

Pre-operative data was acquired to construct the RV/LV/Patch model. The complete LV/RV/Patch FSI model was solved by a commercial finite-element package ADINA (ADINA R & D, Inc., Watertown, MA) using unstructured finite elements and the Newton-Raphson iteration method to obtain full 3D flow, deformation and stress-strain distributions which served as the basis for our investigations. Nonlinear incremental iterative procedures were used to handle fluid-structure interactions. CMR-measured RV volume, pressure and flow data were used to adjust model parameters so that model-predicted RV volume matched CMR-measure data. The RV morphology was modified by trimming the scar and using

different patches to make new models (virtual surgery). The new models were solved again and results were compared with post-operative results for comparisons and validation.

5. Results

5.1 Results from a healthy RV case using the passive model

We started from the healthy ventricle case (given in Fig. 2) using the passive isotropic model to gain some baseline information [7]. Figure 8 gives plots of maximum principal stress (Stress- P_1) and maximum principal strain (Strain- P_1) with a cut-surface chosen so that stress/strain behaviors on the RV inner surface could be observed. Maximum Stress- P_1 /Strain- P_1 values were observed near the RV outlet where RV curvature was large. Figure 9 gives four flow velocity plots at different filling and ejection times showing interesting patterns with multiple vortices. Influence of structural stress/strain and flow shear stress on biological processes involved in ventricle remodeling and recovery after surgery is largely unknown. The implications of these complex flow velocity and shear stress patterns on RV cardiac functions and disease development will be investigated when more patient data become available.

5.2 A patient-specific Tetralogy of Fallot RV model based on pre-operation data

With experience learned from the healthy case, a patient-specific RV/LV/Patch model based on pre-operation data (given in Figure 3) was constructed and solved by ADINA. The model was validated by CMR RV volume data as shown by Fig. 5(d). Figure 10 gives a cut-surface and selected stress/strain plots obtained from the active anisotropic two-layer model. Maximum stress values were found to be closely related to RV curvature and wall thickness, less influenced by stiffness variations. Maximum principal strain (Strain- P_1) distributions demonstrated closer link to stiffness changes, as shown by Figures 10(c) and (e). Fig. 11 gives selected Stress- P_1 and Strain- P_1 plots from the passive isotropic and anisotropic models. Maximum Stress- P_1 values corresponding to maximum RV pressure from the passive isotropic, passive anisotropic, and active anisotropic models were 359.8, 898.1, and 977.3 KPa respectively. The stress values from passive and active anisotropic models were 149.6% and 171.6% higher than that from the isotropic model, respectively. This supports the use of the anisotropic models which give more accurate stress predictions.

5.3 Patch design and virtual surgery, RV cardiac function assessment

With the model tuned using the patient-specific pre-operation material and pressure data (the volume agreement was considered as validation by the pre-operation data), we were ready to perform the virtual surgery, i.e., make new patch models to evaluate different patch design and surgical options. Figure 12 gives sketches of three models: the pre-operation model (M1), patch model 1 (M2, conventional), and patch model 2 (M3, small patch with aggressive trimming). To assess RV cardiac functions and evaluate possible outcome of different surgical options, two commonly-used measures of RV functions were used:

$$\text{Stroke Volume (SV)} = \text{RVEDV} - \text{RVESV}, \quad (8)$$

$$\text{Ejection Fraction (EF)} = (\text{RVEDV} - \text{RVESV}) / \text{RVEDV}, \quad (9)$$

where RVEDV= RV end of diastole volume, RVESV= RV end of systole volume, respectively. RVEDV, RVESV, SV and EF values for the three models are summarized in

Table 1 which indicates that the proposed small patch design would provide about 10% improvement in EF compared to the conventional patch model (41.9% vs. 38.0%).

5.4 Localized stress/strain analysis and tracking method

Figures 10–11 indicated that global maximum stress/strain values often occur at locations with large curvatures, therefore, may not be good indicators for our model/patch comparison and optimization purpose. Our experiences suggest that stress/strain variations at selected locations near the patch may be more sensitive indicators for our model comparisons. Figure 13 gives Stress- P_1 /Strain- P_1 variations tracked at selected locations in the patch area for the three cases showing that stress/strain levels around the patch are considerably lower (50% lower for stress, 40% lower for strain) from the small patch model compared to large patch models. Reduced stress/strain levels around the patch may be desirable for improved RV function and to prevent scarring of peri-patch myocardium, and should be taken into consideration in patch design process. The significance of this finding needs to be supported by further clinical studies.

5.5 Validation using post-operation data and Model Improvement

It should be kept in mind that actual post-operative ventricle morphology, flow and pressure conditions are affected by many complex biological remodeling processes and are not known *a priori*. One important step of our approach was to use actual post-operation data to validate computational predictions and, when needed, adjust the computational model and pressure conditions to better match post-operation CMR measurements. In particular, it was found the post-operative ventricle pressure profile was different and the peak value was about 15% lower than the pre-operative value for the case studied (Fig. 14). The error margin of the first volume prediction was 9.4% as shown by Fig. 14 (c). Noticing the differences in the volume profiles and that it is the same patient with the same ventricle, we modified the prescribed pressure profile and kept the material parameters unchanged. Figure 14 shows that the modified model predicted RV volume with much improved accuracy (new predicted volume 188.76, error < 0.01%; error for the entire cycle < 2%). This modification technique will be applied to future cases so that the first-time prediction accuracy can be improved.

6. Future improvements and directions

Several improvements can be added to our models in the future for better accuracy and applicability: a) Valve mechanics. Valves can be added into our model for better flow control at the inlet and outlet. With that, pulmonary regurgitation can be assessed which is an important part of ToF surgical management; b) Fiber orientation and patient-specific anisotropic material properties. MRI tagging could provide some potential for RV strain field measurement which may help to infer material properties [47]. c) Active contraction model. One way to add active contraction into our model is to introduce an external force field which is tied to fiber structure and orientations. These improvements will be our future directions.

7. Conclusion

Computer-aided surgical design and optimization are still at very early stage. Our preliminary data indicates that MRI-based FSI RV/LV/Patch models validated by pre-operation data have great potential to make accurate assessment of RV cardiac functions and may be used to replace expensive and risky surgical experiments. PVR surgical procedure with a smaller patch and more aggressive scar removal may lead to reduced stress/strain conditions in the patch area and improved recovery of RV functions. Integration of surgical,

imaging, and modeling components could lead to considerable potential gain in not only surgical planning and patient outcome assessment, but also in our understanding of the mechanisms involved in RV failure.

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Figure 1.

A hypothesis that smaller patch and removal of RV scar may lead to improved surgical outcome after surgery. (a) Repaired Tetralogy of Fallot heart with RV outflow patch and scar on anterior RV wall. (b) RV after pulmonary valve insertion (PVR) and reduction of RV outflow patch (*current practice*). (c) RV after pulmonary valve insertion (PVR), reduction of RV outflow patch and removal of RV scar.

Figure 2.

Basic plots showing the modeling procedure using data from a healthy volunteer. (a) A human heart sketch showing left and right ventricles with valve positions; (b) segmented RV MRI contour plots; (c) RV/LV geometry from MRI; (d) the re-constructed 3D geometry of the RV/LV combination model.

Figure 3.
Pre-operation CMR images (end-systole) acquired from a ToF patient and segmented RV/
LV contours.

Figure 4.
Re-constructed 3D geometry of RV and LV from Fig. 5 showing valve and patch locations.

(a) Recorded Tricuspid Valve Pressure

(b) Recorded Pulmonary Valve Pressure

(c) Numerical Valve Pressure Conditions

(d) Patient-Specific Pressure Conditions

Figure 5.

Recorded and prescribed pressure conditions at the tricuspid (inlet) and pulmonary (outlet) valves. Prescribed numerical pressure conditions and valve close/open times were modified from the recorded data so that pressure conditions were as consistent with the recorded data as possible. The vertical bars in (c) indicate valve open/close switch time.

Figure 6.

Stress-Stretch curves from isotropic and passive and active anisotropic models used in this paper. Model parameter values were selected to match CMR pressure-volume data: Isotropic RV tissue: $c_1=7.36$ KPa, $c_2=0$, $D_1=2.88$ KPa, $D_2=4.0$; Scar: $c_1=73.6$ KPa, $c_2=0$, $D_1=28.8$ KPa, $D_2=4.0$; Patch: $c_1=147.2$ kPa, $c_2=0$, $D_1=57.6$ kPa, $D_2=4.0$; Anisotropic Continuity model [22] parameters: $C=18.04$ KPa, $b_1=8.79$, $b_2=1.70$, $b_3=0.774$; (b) End-systolic and end-diastolic Tff and Tcc plots from proposed active patient-specific RV/LV model. End-systolic model parameters: $C=72.16$ KPa, $b_1=8.79$, $b_2=1.70$, $b_3=0.774$; End-diastolic model parameters: $C=22.55$ KPa, $b_1=8.79$, $b_2=1.70$, $b_3=0.774$. (c) RV Volume from Passive Model Compared with CMR Data; (d) RV Volume from Active Model Compared with CMR Data.

Figure 7.

Fiber orientations from a pig model [40], a ToF patient [46], and our patient-specific model. Fiber orientations are illustrated (a) on the epicardium, pig, (b) on the endocardial surfaces. LV fiber orientation is approximately -60° (relative to the circumferential direction) at the epicardium, and $+80^\circ$ at the endocardium. (c) Human ventricle fiber orientation from a patient; (d)–(e) Fiber orientation from our proposed RV/LV model based on patient-specific RV/LV morphologies. RV fiber orientation was set -45° at the epicardium, and $+40^\circ$ at the endocardium. The angles can be adjusted to fit patient-specific data. (f) Two-layer model construction illustration using two slices.

Figure 8.

Stress- P_1 and Strain- P_1 distributions in the RV may provide useful information for mechanical analysis and disease state assessment. (a) 3D view of Stress- P_1 under maximum pressure and position of the cut; (b) Stress- P_1 on the inner surface of RV under maximum pressure (horizontal flipped for better view); (c) Strain- P_1 on the inner surface of RV under maximum pressure; (d) Strain- P_1 on the out-surface of the whole model.

Figure 9.

Velocity plots at different phases showing interesting flow patterns: (a) beginning of the filling phase; (b) flow patterns just before the ending of filling phase; (c) beginning of the ejection phase; (d) ejection continues. Different scale was used for each plot to show flow details.

Figure 10.

A selected cut-surface and Stress- P_1 and Strain- P_1 plots from the active anisotropic ToF patient model at P_{max} (end-diastolic) and P_{min} (end-systolic) conditions.

Figure 11.
Model comparisons: selected Stress- P_1 and Strain- P_1 plots from passive isotropic and passive anisotropic models for the ToF patient.

Figure 12. Sketch of models with different patch designs. (a) Pre-operation model with the old patch and scar tissues; (b) Patch Model 1 with a conventional patch and minimum scar tissue trimming; (c) Patch Model 2 with a small patch and aggressive scar tissue trimming.

(a) Pre-op (Large Patch) Model.

(b) Patch Model 1.

(c) Patch Model 2.

Figure 13.

Stress- P_1 /Strain- P_1 variations tracked at selected tracking points (marked on Strain- P_1 plots) locations for the three cases show that stress/strain levels (around the patches) are considerably lower (>50% for stress, >20% for strain) from Patch Model 2 to the other two models. (a)–(c) Strain- P_1 distributions from the three models are used to show locations of tracking sites. Selected tracking points and marking symbols in the plots: X1: *, just below the patch (or scar for M2); X2: x, just next to the left of the patch; X3: o, just above the patch; X4: +, just next to the right of the patch; X5: ∇, at the center of the patch; X6: Δ, just below the patch (this is for pre-op model only).

(a) Post-Op CMR Images (10 out of 12) from the Same Patient.

(b) Numerical Pre- and Post-Op Pressure Conditions.

(c) Model-Predicted and CMR-Measured RV Volumes.

Figure 14.

Validation by post-operative data. (a) Post-operative CMR images; (b) Computational pre- and post-operative pressure conditions in RV; (c) Comparison between measured RV volume and two computational predictions. Blue line: volume using pre-operative pressure (CMR max volume 188.3 ml, predicted volume 216.4, error 14.9%); black line: volume using adjusted pressure condition; red asterisk *: CMR-measured post-operative RV volume (new predicted volume 188.76, error < 0.01%; error for the entire cycle < 2%).

Diagram 1.

Three groups of data for computational ventricle modeling.

Diagram 2.

Flowchart of ventricle modeling for surgical planning and optimization.

Table 1

Summary of stroke volume and ejection fraction from 3 patch models showing that the small patch model improved ejection fraction (EF) by about 10% compared to the conventional procedures.

	Pre-Op Model	Patch Model 1	Patch Model 2	CMR-Data
	Old (large) Patch + Scar	New Model Large Patch	New Model Small Patch	Post-Op actual data
RVEDV (ml)	409.2	265.9	216.4	406.9
RVESV (ml)	254.3	164.8	125.8	254.5
SV	154.9	101.1	90.6	73.3
EF (%)	37.9	38.0	41.9	37.45