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Multi-Physics MRI-Based Two-Layer Fluid-Structure Interaction Anisotropic Models of Human Right and Left Ventricles with Different Patch Materials: Cardiac Function Assessment and Mechanical Stress Analysis

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Abstract

Multi-physics right and left ventricle (RV/LV) fluid-structure interaction (FSI) models were introduced to perform mechanical stress analysis and evaluate the effect of patch materials on RV function. The FSI models included three different patch materials (Dacron scaffold, treated pericardium, and contracting myocardium), two-layer construction, fiber orientation, and active anisotropic material properties. The models were constructed based on cardiac magnetic resonance (CMR) images acquired from a patient with severe RV dilatation and solved by ADINA. Our results indicate that the patch model with contracting myocardium leads to decreased stress level in the patch area, improved RV function and patch area contractility.

Keywords

Right ventricle; congenital heart disease; heart model; Dacron scaffold patch; fluid-structural interaction

1 Introduction

Recent advances in computational modeling, methods and computer technology have made it possible for computer-simulated procedures to be used in clinical decision-making process to replace empirical and often risky clinical experimentation to examine the efficiency and suitability of various reconstructive procedures and patch design in diseased hearts [1-18]. A large amount of effort has been devoted to quantifying heart tissue mechanical properties

and fiber orientations mostly using animal models [16,19-23]. Humphrey's book provided a comprehensive review of the literature [24]. 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 [25-27]. Bathe, Hou, and Rugonyi et al. made considerable advances for models and methods with fluid-structure interactions, implementation of finite element procedures and application of the methods to many realistic engineering and biological problems [28-36]. Their models, methods and finite element procedures contributions were summarized in [28,30-31].

Right ventricular (RV) dysfunction is a common cause of heart failure in patients with congenital heart defects and often leads to impaired functional capacity and premature death [37]. 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. The current surgical approach, which includes pulmonary valve replacement/insertion (PVR), has yielded mixed results [38-40]. New surgical options including scar tissue reduction and RV remodeling have been proposed in order to improve RV function recovery [37]. The RV remodeling aims mainly to reduce RV volume by removing or reducing the non-contracting tissue (scar and patch) in the outflow area of the RV. This scar and patch tissue is placed at the original operation to enlarge the very narrow RV outflow and pulmonary valve. Over time, this tissue often stretches and can become quite large. As part of the second procedure, excess scar and the original patch are removed and a new smaller patch is placed in the RV outflow, along with pulmonary valve insertion.

In our previous papers, computational right and left ventricle (RV/LV) models with fluid-structure interactions were introduced to assess outcomes of various RV reconstruction techniques with different scar tissue trimming and patch sizes [1-3]. For patch materials, myocardial tissue regeneration techniques are being developed for the potential that viable myocardium may be regenerated or placed in the patch area [41-42]. Wald and Geva et al. (2009) investigated effects of regional dysfunction on global RV function in patients with repaired ToF and reported that localized dysfunction in the region of the RV outflow tract patch adversely affects global RV function and regional measures, and may have important implications for patient management [43].

In this paper, RV/LV/Patch models with fluid-structure interactions (FSI) based on patient-specific cardiac magnetic resonance (CMR) imaging data were constructed to investigate the effects of patch material mechanical properties on RV cardiac function and mechanical performances. The models included (a) fluid-structure interaction for the right ventricle; (b) two-layer ventricle wall construction with realistic epicardium and endocardium fiber orientations; (c) active anisotropic material properties; (d) a patch area with different materials; (e) a structure-only LV as support to the RV structure. Active ventricular contraction was modeled by a material stiffening approach. Three patch materials were considered: Patch 1 - Dacron scaffold; Patch 2 - pericardium, usually from the patient, treated with glutaraldehyde to crosslink the collagen and make it stiffer; Patch 3 - viable contracting myocardium (not currently available but represents future direction). The 3D CMR-based RV/LV/Patch FSI models were solved to obtain 3D ventricular deformation, local patch area contraction ratio, and stress/strain distributions for accurate assessment of RV cardiac function and mechanical conditions. The computational models were validated by CMR data and then used to assess the effect of patch material properties with the ultimate goal of improving recovery of RV function after surgery.

2. Data Acquisition, Models and Methods

2.1 Data acquisition

Cardiac MRI (CMR) studies were performed by Dr. Tal Geva to acquire patient-specific ventricular geometry for patients needing RV remodeling and pulmonary valve replacement operations before and after scheduled surgeries (Fig. 1). The RV and LV were imaged using ECG-gated, breath-hold steady state free precession cine MR in the ventricular short axis (12-14 equidistant slices covering the ventricles from base to apex; slice thickness 6-8 mm; interslice gap 0-2 mm; 30 frames per cardiac cycle). The valve and patch positions were determined with cine MR imaging, flow data, and delayed enhancement CMR to delineate location and extent of scar/patch. The CMR findings were subsequently confirmed by the intra-operative observations (del Nido). Three dimensional RV/LV geometry and computational mesh were constructed following the procedures described in [1-3]. Fig. 1 shows one set of pre-operative CMR images from a patient with repaired TOF and severe RV dilatation. The figure depicts segmented contours for both tissue and scaffold patches. Fig. 2 shows the stacked contours, RV/LV inner/outer surfaces, valve and patch positions. Figure 3 shows patch thickness using a selected cut surface. Figure 4 shows ventricular fiber orientations on epicardium and endocardium from human and a pig model and how the two-layer RV/LV model was constructed [15,22].

2.2 The fluid model

Blood flow in the right ventricle was assumed to be laminar, Newtonian, viscous and incompressible. The Navier-Stokes equations with arbitrary Lagrangian-Eulerian (ALE) formulation were used as the governing equations. To simplify the computational model, the cardiac cycle was split into two phases: **a)** the filling phase (diastole) when the inlet was open, inlet blood pressure was prescribed (Fig. 5), blood flows into the RV, and the outlet was closed (by setting flow velocity to zero); **b)** The ejection phase (systole) when inlet was closed, outlet was open, outlet pressure was prescribed, and blood was ejected out of the RV. Pressure conditions were prescribed at the tricuspid (inlet) and pulmonary (outlet) valves (see Fig. 5 and [1,44]). RV tissue material parameters (see next section) were modified so that the RV would contract and expand properly and match the CMR-measured RV volume data (Fig. 5). 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 [1-3,28,30-31]. The fluid model is given below ($i=1,2,3$, summation convention was used):

$$\rho \left(u_{i,t} + (u - u_g)_j u_{i,j} \right) = - p_{,i} + \mu u_{i,jj}, \quad (1)$$

$$u_{j,j} = 0, \quad (2)$$

$$u_i | \Gamma = x_{i,t}, \quad (3)$$

$$p|_{\text{inlet}} = p_{in}(t), \text{ (inlet open)}, u_i|_{\text{inlet}} = 0, \text{ (inlet closed)}, \quad (4)$$

$$P|_{\text{outlet}} = p_{\text{out}}(t), (\text{outlet open}), u_i|_{\text{outlet}} = 0, (\text{outlet closed}), \quad (5)$$

$$p|_{\text{LV}} = p_{\text{LV}}(t), \quad (6)$$

$$\sigma_{ij}^r n_j^r|_{\text{interface}} = \sigma_{ij}^s n_j^s|_{\text{interface}}, \quad (7)$$

where $\mathbf{u}$ and p are fluid velocity and pressure, $\mathbf{u}_g$ is mesh velocity, Γ stands for RV inner wall, $f_{\bullet,j}$ stands for derivative of f with respect to the j th variable (or time t), σ^r and σ^s are fluid and structure stress tensors, and $\mathbf{n}^r$ and $\mathbf{n}^s$ are their outward normal directions, respectively.

2.3 The active anisotropic solid models, two-layer model construction

The RV and LV materials were assumed to be hyperelastic, anisotropic, nearly-incompressible and homogeneous. Patch materials (other than Patch 3 which was assumed to be normal contracting myocardium) were assumed to be hyperelastic, isotropic, nearly-incompressible and homogeneous. The governing equations for the structure models are:

$$\rho \quad v_{i,tt} = \sigma_{ij,j}, \quad i, j = 1, 2, 3; \text{sum over } j, \quad (8)$$

$$\varepsilon_{ij} = (v_{i,j} + v_{j,i} + v_{\alpha,i} \quad v_{\alpha,j}) / 2, \quad i, j, \alpha = 1, 2, 3, \quad (9)$$

where σ is the stress tensor, ε is the strain tensor, $\mathbf{v}$ is displacement, $f_{\bullet,j}$ stands for derivative of f with respect to the j th variable, and ρ is material density. Equations (8)-(9) were used for RV/LV tissue and patch materials. The normal stress was assumed a) to be zero on the outer RV/LV surface; b) to be equal to the normal stress imposed by fluid forces on the inner RV surface as specified by Eq. (7); and c) to be equal to the normal stress corresponding to the LV pressure as specified by Eq. (6) on the LV inner surface. The left ventricle was included in the RV/LV/Patch model as a structure-only model to provide structural support to the right ventricle so that ventricular deformation and stress/strain distribution would be reasonable. Structure-only LV model was used to save model construction effort and computing time. Without the LV support, other artificial boundary conditions would need to be added to keep the wall between RV and LV in the right position and shape. Those artificial boundary conditions would be hard to set and justify.

The nonlinear Mooney-Rivlin model was used to describe the nonlinear anisotropic and isotropic material properties. The strain energy function for the isotropic modified Mooney-Rivlin model is given by [1,28,30]:

$$W = c_1 (I_1 - 3) + c_2 (I_2 - 3) + D_1 [\exp(D_2 (I_1 - 3)) - 1], \quad (10)$$

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

$$I_1 = \Sigma C_{ii}, I_2 = 1/2 [I_1^2 - C_{ij}C_{ij}], \quad (11)$$

$C = [C_{ij}] = X^T X$ is the right Cauchy-Green deformation tensor, $X = [X_{ij}] = [\partial x_i / \partial a_j]$, (x_i) is the current position, (a_i) is the original position, c_i and D_i are material parameters chosen to match experimental measurements [4,21,24]. The strain energy function for the anisotropic modified Mooney-Rivlin model anisotropic model was obtained by adding an additional anisotropic term in Eq. (10) [28, 45]:

$$W = c_1 (I_1 - 3) + c_2 (I_2 - 3) + D_1 [\exp(D_2 (I_1 - 3)) - 1] + K_1 / (2K_2) \exp[K_2 (I_4 - 1)^2 - 1], \quad (12)$$

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 [28]. A two-step least-squares method was used to determine the parameter values in (12) to fit the experimental data given in [4-5]. Step 1: With D_2 and K_2 fixed, least square approximation technique was used to obtain C_1 , D_1 , K_1 (all dependent on D_2 and K_2) so that the stress-strain curves in the fiber and circumferential directions derived from (12) have minimum error (best match) with experimental data. Step 2: Let D_2 and K_2 change from -100 to 100 , we perform Step 1 to get the corresponding C_1 , D_1 , K_1 values, and the fitting error for all (D_2, K_2) combinations with initial increment=10. Optimal (D_2, K_2) and the associated C_1 , D_1 , K_1 values are determined by choosing the pair corresponding to a minimum error. Searching increment for (D_2, K_2) starts from 10 for $[-100, 100]$ and then refines to 1, and 0.1 when the search domain is reduced. Even though the procedure involves huge number of calculations, it is fully automatic and can be used to find the best fit for any measured experimental data. Choosing $c_1=0.351$ KPa, $c_2=0$, $D_1=0.0633$ KPa, $D_2=5.3$, $K_1=1.913$ KPa, $K_2=6.00$, it was shown [1-3] that stress-strain curves derived from our model (12) agrees very well with the stress-strain curves from the dog model given in [4-5]. The stress-stretch curves of scaffold patch, treated pericardium and normal myocardium are given in Fig. 6. The Young's modulus for the Dacron scaffold patch was measured to be 80 MPa and parameter values in the Mooney-Rivlin model were chosen to match the linear model.

Modeling active heart contraction is much harder because stress in a pumping human heart cannot be measured in vivo non-invasively. Since it is difficult 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 will contract/relax by following a time-dependent stiffening/relaxation material model. The time-dependent material parameters in (12) (all parameters were functions of time) were numerically determined to match the CMR measured RV volume curve (see Fig. 5). The material parameter adjustment processes for both RV and LV were the same. Parameter values in the Mooney-Rivlin models for all materials are summarized in Table 1. c_2 was set to zero in all models.

As patient-specific fiber orientation data was not available in practice, we chose to construct a two-layer RV/LV model and set fiber orientation angles using fiber angles given in Hunter [15] (also see Fig. 4). Fiber orientation can be adjusted when patient-specific data becomes available [22].

2.4 A pre-shrink process for in vivo CMR-based models and geometry-fitting mesh generation technique

It is normally easier that the numerical procedure for the computational model starts from geometries with zero flow velocity, zero pressure and zero stress/strain distributions since the initial flow velocity, pressure and ventricle stress/strain conditions of a working heart required by the initial-boundary value RV/LV FSI model are near impossible to measure. Under the *in vivo* condition, the ventricles were pressurized and the zero-stress ventricular geometries were not known. In our model construction process, a pre-shrink process was applied to the in vivo end-systolic ventricular geometries to generate the starting shape (zero ventricle pressure) for the computational simulation. The initial shrinkage for the inner ventricular surface was 2-3% and end-systolic pressure was applied so that the ventricles would regain its *in vivo* morphology. The out surface of the ventricular shrinkage was determined by conservation of mass so that the total ventricular wall mass was conserved. Without this shrinking process, the actual computing domain would be greater than the actual ventricle due to the initial expansion when pressure was applied.

Because ventricles have complex irregular geometries with patch and scar tissue component inclusions which are challenging for mesh generation, a geometry-fitting mesh generation technique was developed to generate mesh for our models. Figure 4(g) gives an illustration of RV/LV geometry between two slices. Each slice was first divided into geometry-fitting areas (called “surfaces” in ADINA). The neighboring slices were stacked to form volumes. Using this technique, the 3D RV/LV domain was divided into many small “volumes” to curve-fit the irregular plaque geometry with patch as an inclusion. 3D surfaces, volumes and computational mesh were made under ADINA computing environment. For the RV/LV/ Patch model (Patch 1) constructed in this paper, the finite element ADINA FSI solid model had 973 small volumes. The fluid part had 272 volumes. Mesh analysis was performed by decreasing mesh size by 10% (in each dimension) until solution differences were less than 2%. The mesh was then chosen for our simulations.

2.5 The solution methods and simulation procedures

Equations (1)-(12) gave the full RV/LV/Patch FSI model which was solved by ADINA (ADINA R&D, Watertown, MA, USA) using unstructured finite elements and the Newton-Raphson iteration method [28,30]. The “Re-Start” feature in ADINA was used to adjust material parameters at each numerical time step to implement the active contracting material properties. With validation from actual RV volume measurements, our simulated RV motion and volume change can provide ventricular cardiac function assessment and flow and stress predictions for detailed mechanical analysis.

3. Results

3.1 Comparison of stress/strain behaviors of the three patch models

Three patch models were constructed and solved for patch comparison and cardiac function analysis: Model 1 (M1) consisted of Dacron scaffold patch; Model 2 (M2) consisted of glutaraldehyde-treated pericardial patch; and Model 3 (M3) consisted of viable contracting myocardium in the patch area. Figure 7 presents maps (band plots) of maximum principal stress (Stress- P_1) from the three models corresponding to maximum (end of diastole) and minimum (end of systole) pressure conditions to give an overall view of the stress distributions. Maximum Stress- P_1 (1004 kPa) from Model 1 was about 60% higher than those from Models 2 & 3. To obtain more specific information caused by the patch differences, 5 locations (X1-X5) were selected and Figures 8-9 show Stress- P_1 and Strain- P_1 (maximum principal strain) tracked at those five locations over a cardiac cycle. Stress- P_1 at X5 (center location) from the Dacron scaffold patch was 404.4 kPa, which was about 350%

and 600% higher than that from M2 (70.3 kPa) and M3 (68.9kPa), respectively. Stress and strain behaviors at the 5 locations can be seen in Figures 8-9 and maximal values are summarized in Table 2. Clearly Model 3 (viable myocardium) showed the lowest overall stress level in the patch area.

3.2 Assessment of RV cardiac function and regional patch area changes

Results from the three patch models were used to evaluate their effect on RV function. The two common measures of RV function evaluated were stroke volume (SV) and ejection fraction (EF) defined as:

$$SV = RV \text{ End Diastolic Volume (RVEDV)} - RV \text{ End Systolic Volume (RVESV)}; \quad (13)$$

$$EF = [RVEDV - RVESV] / RVEDV; \quad (14)$$

RVEDV, RVESV, SV and EF values for M1, M2, and M3 are summarized in Table 3. The results indicate that Patch Model 3 (M3) would provide about 3.8% improvement in EF (absolute EF improvement was 1.4%), compared to Patch Model 1 (Dacron scaffold). The improvement was relatively small because the patch area was only a small portion of the right ventricle (3%). The observation that EF values for M1 and M2 were very close can be attributed to the fact that both patch materials were considerably stiffer than normal myocardium and that the total patch area was only a small portion (3%) of the total RV surface area (end-diastolic RV surface area = 320 cm²).

To concentrate on the patch area and illustrate the differences among the three patch materials, patch area changes are summarized in Table 4 where

$$RVEDPA = RV \text{ End Diastolic Patch Area}, \quad (15)$$

$$RVESPA = RV \text{ End Systolic Patch Area}. \quad (16)$$

Patch 3 had an impressive 27.6% patch fractional area change, a regional tissue contracting measure similar to ejection fraction for the whole ventricle. Dacron patch area hardly changed (fractional area change was only 0.44%) because the scaffold material was very stiff. The pericardium patch area changed about 7%, better than the Dacron patch. Fig. 10 shows the area variations of the three patches over the cardiac cycle.

3.3 Flow velocity has complex patterns in the ventricle

Figure 11 shows the plots of the velocity patterns within a cardiac cycle. Figures 11 (a)-(f) used one uniform scale, showing relative velocity magnitude at different time points in a cardiac cycle. Figures 11 (g) and (h) are re-plot of (a) and (f) using a scale for each individual plot so that flow patterns becomes visible. During the filling phase, blood enters the ventricle from the tricuspid valve, the pulmonary valve is closed, and the right ventricle relaxes at the same time. Fig. 11(d) shows the beginning of the ejection phase when the ventricle starts to contract, the tricuspid valve is closed, and blood is ejected out through the pulmonary valve. Table 5 gives the peak velocity values at the tricuspid and pulmonary valves at selected time points from the three models. Flow velocity and shear stress

information will be useful for detailed mechanical analysis for potential disease development and tissue engineering when local flow environment becomes relevant.

4. Discussion

4.1. Motivation to develop myocardium tissue regeneration techniques

With the rapidly increasing number of late survivors of Tetralogy of Fallot repair, surgical management of patients with right ventricular dysfunction has become a major clinical challenge. The wide variability in clinical status, extent of right ventricular dilatation, scarring, and dysfunction at the time of presentation has resulted in disparate surgical results with pulmonary valve insertion alone [40]. Del Nido and Geva et al. have proposed aggressive scar tissue trimming and RV volume remodeling as a way to improve RV function after pulmonary valve replacement surgery [37]. Detailed description of the surgical procedures is beyond the scope of this modeling paper. Very briefly, the surgeon sews the patch in the ventricular wall after the man-made valve is placed between ventricle and the pulmonary artery. Location of the patch can be seen from Figures 2 & 3. The patch is normally passive and just moves with heart beat due to the internal load placed on it by the pressure developed in the ventricle. Recent work suggests that contractile cells can be generated, providing a contracting scaffold [41-42]. If active tissue can be used, the patch would be able to contract, like normal heart tissue. That will improve cardiac function. That is the motivation of the study. Results presented in this paper indicate that tissue regeneration techniques that restore RV myocardium have the potential to improve ventricular function. Computational simulations may be used to supplement/replace empirical and often risky clinical experimentation, or even guide the design of new clinical trials to examine the efficiency and suitability of various reconstructive procedures in diseased hearts.

4.2. Mechanical analysis and its relevance to ventricular remodeling, myocardial regeneration, and surgical design

It is well known that mechanical forces play an important role in biological processes. Hunter, McCulloch, Guccione and other authors have made considerable contributions for ventricle models which serve as foundation for many further development and investigations [4-20,22-27]. The *in vivo* MRI-based human RV/LV/Patch model with fluid-structure interactions is an improvement over models found in the current literature. The FSI model allows us to investigate both flow and structure stress/strain behaviors and their influences on various biological processes, including ventricle remodeling and myocardium regeneration. As we continue our investigations on ventricular remodeling after pulmonary valve replacement surgery and myocardium tissue regeneration following stem cell seeding, detailed localized mechanical stress and flow conditions will be obtained to quantify their effects and influence on ventricular remodeling, myocardium tissue regeneration, and related cellular activities. It should be noted that it is not possible to simulate blood flow in the ventricle using rigid wall models. Ventricular contraction and deformation (with volume variation nearly 100%) are the main contributors to blood flow in ventricles. Without ventricular deformation, with the pulmonary (or tricuspid) valve closed, blood cannot even flow into (or eject out of) the ventricle. Our multi-physics RV/LV/Patch FSI model can serve as a useful tool to investigate cellular biology and tissue regeneration under localized flow and structural stress environment.

4.3 Model limitations

Several improvements can be added to our models in the future for better accuracy and applicability: a) direct measurements of tissue mechanical properties which will be very desirable for improved accuracy of our models; b) inclusion of pulmonary valve mechanics

in the model to simulate regurgitation; c) multi-scale models including organ, cell, and gene investigations.

5. Conclusion

The CMR-based multi-physics RV/LV/Patch FSI model introduced in this paper provides more accurate ventricle volume and mechanical stress predictions compared to previous models [1-3] and can be used as a tool for surgical planning and mechanical analysis for ventricular remodeling and tissue regeneration investigations. Our preliminary results indicate that a patch model using viable myocardium leads to decreased stress level in the patch area, improved right ventricular function, and higher patch area contractility. Maximum Stress- P_1 value at the center of the patch from the Dacron scaffold patch model was 350%-600% higher than that from the other two models. Patch area reduction ratio was 0.44%, 7.3% and 27.6% from Dacron scaffold patch, treated pericardium patch, and viable contracting myocardial patch, respectively. The techniques to regenerate myocardium combined with other surgical procedures have promising potential to improve ventricular function in patients with repaired TOF and other congenital heart disease.

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Figure 1.

Pre-operative CMR images (end-systole) acquired from a patient, segmented RV/LV contours, and smoothed contours for 3D model construction. (a) CMR images from a patient; (b) segmented RV/LV contours; (c) smoothed contours; (d) modified contours with Dacron patch showing patch thickness.

Figure 2.
Re-constructed 3D geometry of RV and LV showing inner and outer surfaces, valve and patch locations.

Figure 3.
A selected cut-surface showing patch thickness and location.

Figure 4. Fiber orientation on epicardium and endocardium from (a)-(b) a pig model [15]; (c)-(d) a human heart [22]; (e)-(f) our RV/LV/Patch model; (g) two-layer model construction.

Figure 5.

Simplified two-phase patient-specific pressure conditions imposed at the inlet and outlet and computed RV volumes compared with CMR recorded data showing good agreement (CMR measured minimum volume = 254.5 ml, maximum volume = 406.9 ml; active model (Model 2) predictions: minimum volume = 254.6 ml, maximum volume = 406.9 ml; error margin <1%).

Figure 6.

Patient-specific material stress-stretch curves for ventricular tissue and patch materials. T_{ff} stands for stress in the fiber direction. T_{cc} stands for stress in transverse direction. Parameter values for all models are summarized in Table 1. (a) Stress-stretch curves from passive isotropic and anisotropic models. (b) End-systolic and end-diastolic T_{ff} and T_{cc} plots from active patient-specific RV/LV model. Only stiffness in the fiber direction was varied during the cardiac cycle.

Figure 7.

Plot of maximal principal stress (Stress- P_1) distributions corresponding to maximum and minimum pressure on the inner RV surfaces from the three patch models showing that patch material has consideration effect on stress patterns and that Dacron scaffold patch model has non-uniform stress/strain distributions. Different color scales were used for the maximum pressure group and minimum pressure group plots (one scale for each group).

Figure 8.

Maximum principal stress (Stress- P_1) variations tracked at selected tracking locations for the three patch models show that stress levels around the patch are considerably lower from Patch Model 3 than that from the other two models. Lower stress levels may indicate that the ventricle operates under more favorable mechanical conditions.

Figure 9.

Maximum principal strain (Stress- P_1) variations tracked at selected tracking locations for the three patch models show that strain levels around the patch are considerably higher from Patch Model 3 than that from the other two models. Higher strain level indicates that regenerated viable myocardium patch (patch 3) is contributing more to cardiac function (pumping blood).

Figure 10.

Patch area variations showing that patch with contracting myocardium material provided larger patch area variations which led to better ventricular ejection fraction. All three patches had about the same end-of-diastole areas. Total patch area is only a small portion (3%) of the total RV surface area (end of diastole RV surface area = 320 cm²).

Figure 11.

Plot of flow velocity patterns in a cardiac cycle from Model 2. Flow shear stress can be calculated for detailed location-specific stress analysis. Flow shear stress may play an important role in the ventricle remodeling and tissue regeneration processes and detailed information can be useful for surgical procedure optimization and patch selection.

Table 1
Summary of parameter values in the modified Mooney-Rivlin models for all materials, isotropic, anisotropic, passive and active models

Material/Model	c_1 (kPa)	D_1 (kPa)	D_2	K_1 (kPa)	K_2
Scaffold Patch (Isotropic)	6000	3000	1.4	0	
Pericardium (Isotropic)	13.26	13.26	9.0	0	
Passive Anisotropic Model					
Passive RV/LV Inner Layer	3.124	0.984	3.0	14.953	3.0
Passive RV/LV Outer Layer	3.566	0.918	3.0	14.532	3.2
Active Model, End of Diastole					
RV/LV Inner Layer	3.280	2.470	2.9	17.742	2.9
RV/LV Outer Layer	0.688	0.028	5.0	37.518	2.7
Active Model, End of Systole					
RV/LV Inner Layer	5.531	3.353	3.0	52.560	2.8
RV/LV Outer Layer	3.525	2.488	3.3	67.395	2.6

Table 2
Summary of maximal values of tracking curves at five locations from the three models

Location	Stress- P_1 (kPa)			Strain- P_1 (kPa)		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
X1(below)	91.7790	96.5068	80.8184	0.3454	0.2318	0.5051
X2(left)	235.0560	70.9693	35.8888	0.2625	0.4945	0.3414
X3(top)	889.1000	223.4365	142.2495	0.3444	0.3046	0.3315
X4(right)	349.3760	42.4289	40.2233	0.2202	0.1615	0.1889
X5(center)	404.4220	89.4236	57.288	0.011	0.0678	0.3734

Table 3

Stroke volume and ejection fraction comparisons for three patch models showing regenerated myocardium model has the best ejection fraction

Cases	Patch Model 1 Dacron Scaffold	Patch Model 2 pericardium	Patch Model 3 Myocardium
RVEDV (ml)	403.8	406.9	415.9
RVESV (ml)	253.7	254.6	255.4
SV (ml)	150.1	152.3	160.5
Ejection Fraction (%)	37.2%	37.4%	38.6%

Table 4

Patch area change comparisons for three patch models showing regenerated myocardium model has the best patch area change ratio

Cases	Patch Model 1 Dacron Scaffold	Patch Model 2 pericardium	Patch Model 3 Myocardium
RVEDPA (cm2)	9.589	9.593	9.320
RVESPA (cm2)	9.547	8.919	6.742
Patch Area Change	0.042	0.674	2.578
Patch Area Change Fraction (%)	0.44%	7.03%	27.6%

Table 5

Peak velocity values at the tricuspid and pulmonary valves for selected time points from the three models

Peak Velocity	Filling, Vmax at Inlet (cm/s)			Ejection, Vmax at outlet (cm/s)		
Time (s)	1.10	1.14	1.24	1.70	1.74	2.16
Model 1	27.84	109.4	127.8	89.36	220.3	27.30
Model 2	23.37	106.0	124.2	92.94	220.4	27.69
Model 3	28.02	110.5	131.6	94.09	224.9	27.56