SURGERY FOR ADULT CARDIOVASCULAR DISEASE

THE EFFECT OF VENTRICULAR VOLUME REDUCTION SURGERY IN THE DILATED, POORLY CONTRACTILE LEFT VENTRICLE: A SIMPLE FINITE ELEMENT ANALYSIS

Mark B. Ratcliffe, MD
James Hong, MS
Ali Salahieh, BS
Stuart Ruch, MD, PhD
Arthur W. Wallace, MD, PhD

Objectives: Ventricular volume reduction surgery has been proposed by Batista to improve cardiac function in patients with dilated cardiomyopathy. However, limited clinical data exist to determine the efficacy of this operation. A finite element simulation is therefore used to determine the effect of volume reduction surgery on left ventricular end-systolic elastance, diastolic compliance, stroke work/end-diastolic volume (preload recruitable stroke work), and stroke work/end-diastolic pressure (Starling) relationships. **Methods:** End-diastole and end-systole were represented by elastic finite element models with different unloaded shapes and nonlinear material properties. End-systolic elastance, diastolic compliance, preload recruitable stroke work, and Starling relationships, as well as energy expenditure per gram of unresected myocardium, were calculated. Two different types of volume reduction surgery (apical and lateral) were simulated at 10% and 20% left ventricular mass reduction. **Results:** Ventricular volume reduction surgery causes diastolic compliance to shift further to the left on the pressure-volume diagram than end-systolic elastance. Volume reduction surgery increases the slope of the preload recruitable stroke work relationship (dilated cardiomyopathy 0.006 J/mL; 20% lateral volume reduction surgery 0.009 J/mL) but decreases the slope of the Starling relationship (dilated cardiomyopathy 0.028 J/mm Hg; 20% lateral volume reduction 0.023 J/mm Hg). For a given amount of resection, lateral volume reduction has a greater effect than apical volume reduction. Ten-percent and 20% lateral volume reduction reduces energy expenditure by 7% and 17%, respectively. **Conclusion:** Ventricular volume reduction surgery shifts end-systolic elastance and diastolic compliance to the left on the pressure-volume diagram. The net effect on ventricular function is mixed. Volume reduction surgery increases the slope of preload recruitable stroke work, but increased diastolic compliance causes a small decrease in the Starling relationship (3 mm Hg difference between dilated cardiomyopathy and volume reduction surgery at stroke work = 0.5 J). (J Thorac Cardiovasc Surg 1998;116:566-77)

From the Division of Cardiothoracic Surgery, Department of Surgery, and the Department of Anesthesia, School of Medicine of the University of California, San Francisco, and the San Francisco Veterans Affairs Medical Center.

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Address for reprints: Mark B. Ratcliffe, MD, 112D, San Francisco Veterans Affairs Medical Center, 4150 Clement St, San Francisco, CA 94121.

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A new surgical therapy for heart failure has recently gained attention.¹⁻⁵ The operation, first performed by a Brazilian surgeon, Randal Batista, is known as ventricular volume reduction surgery (VVRS) and includes the resection of a viable "slice" of the lateral left ventricular (LV) wall in patients with dilated cardiomyopathy (DCM) and end-stage congestive heart failure. Although early results with this procedure in uncontrolled trials have been promising, experimental analysis of the effect of the Batista operation on LV mechanics is limited.⁶⁻¹⁰

The success of an operation that surgically remodels ventricular size, shape, or regional stiffness depends on how the procedure affects *both* end-systolic and end-diastolic pressure-volume relationships and how those changes affect ventricular function (stroke-work/end-diastolic volume; preload recruitable stroke work) [PRSW],¹¹ and stroke-work/end-diastolic pressure relationships [Starling].¹² End-systolic and end-diastolic pressure-volume relationships, respectively termed end-systolic elastance and diastolic compliance, are mechanically determined by respective LV regional material properties (stiffness) and unloaded ventricular shapes.¹³ VVRS will change the unloaded end-systolic and end-diastolic ventricular shapes and regional stiffness in and around the surgical repair site. As a consequence, postoperative ventricular elastance and compliance are altered and may shift by different absolute amounts. As a result, the stroke work/end-diastolic volume and stroke work/end-diastolic pressure relationships may shift. At present, the effect of VVRS on systolic elastance, diastolic compliance, and the resultant net effect on ventricular function is unknown.

Finite element models relate regional material properties, structural shape, and regional stress as a system of linear equations.¹⁴ Typically a structure, in this case the LV, is composed of "elements," material properties (regional stiffness) are assigned to each element, external loads are applied (intracavitary pressure), and regional stresses and structural deformation are calculated. In this study, we describe a finite element model of the dilated LV with reduced contractility in which the effects of partial apical and lateral wall ventricular resections are simulated.

The primary goal of this study is to model the effect of VVRS on end-systolic elastance, diastolic compliance, and ventricular function (stroke work/end-diastolic volume and stroke work/end-diastolic pressure [Starling] relationships). We also test the hypothesis that 2 different types of partial ventriculectomy involving either the lateral wall or apex had different effects on elastance, compliance, and ventricular function. Finally, we test the hypothesis that VVRS decreases myocardial energy expenditure.

Methods

Overview of finite element model. A finite element model of the dilated poorly contractile LV was developed. Diastole and end-systole were represented by separate elastic models that have different unloaded shapes and material properties (Fig 1, step 1). Two types of VVRS simulations (apical VVRS and lateral VVRS) were developed (Fig 1, steps 3 and 4). Finite element meshes were created and loaded with a range of physiologic intraventricular pressures with the use of commercially available computer-assisted design and simulation

Fig 1. Schematic of modeling process used in this study. Step 1: The unloaded diastolic LV is modeled as a thick-walled axisymmetric hemi-ellipsoid based on images of dilated human cardiomyopathic hearts. The unloaded systolic LV shape was derived from the diastolic shape. In this and subsequent steps only the right half of the axisymmetric shape is shown. Step 2: Material properties and boundary conditions are assigned. The base of the LV is fixed in both the x and y directions while the apex is fixed in the x direction only (hatched arrows). In this and the subsequent step the pre-resection shape is seen as the dashed line. Step 3: Apical VVRS: Apical resection is performed by reducing the long-axis radii, $a1$ and $a2$, while maintaining a constant wall thickness. The short-axis radii, $b1$ and $b2$, are unchanged. Step 4: Lateral VVRS: Lateral resection is performed by reducing the short-axis radii, $b1$ and $b2$, while maintaining a constant wall thickness. The long-axis radii, $a1$ and $a2$, are unchanged.

software (Ideas, release 2.0, SDRC, Inc, Milford, Ohio). Nonlinear material properties were assigned and the models solved with the use of a commercially available nonlinear finite element solver (ABAQUS, Standard module, version 5.4, Hibbet, Karlson and Sorenson, Inc, Providence, RI). Finite element modeling and analysis were performed on a Unix-based workstation (SPARCsystem 10, model 40, Sun Microsystems, Inc, Mountain View, Calif).

Structure.

Initial unloaded diastolic shape. The unloaded dilated LV shape is a thick-walled axisymmetric hemi-ellipsoid. Diastolic dimensions of the unloaded dilated LV were chosen empirically so that when the diastolic model was loaded with an intracavitary LV pressure of 20 mm Hg, LV dimensions approximated dimensions from patients with DCM.¹⁵⁻¹⁸

Initial unloaded end-systolic shape. The unloaded end-systolic LV shape was derived from the diastolic shape by assuming that LV wall volume and aspect ratio are constant and that average circumferential and longitudinal strains are equal to the strain of an unloaded sarcomere during contraction. End-systolic dimensions were calculated by simultaneously solving the following 4 equations (Mathematica, version 2.2, Wolfram Research Inc, Champaign, Ill):

$$[b_{2,ES}^2 a_{2,ES} - b_{1,ES}^2 a_{1,ES}] = [b_{2,ED}^2 a_{2,ED} - b_{1,ED}^2 a_{1,ED}] \quad (1)$$

$$\frac{a_{2,ES} - a_{1,ES}}{b_{2,ES} - b_{1,ES}} = \frac{a_{2,ED} - a_{1,ED}}{b_{2,ED} - b_{1,ED}} \quad (2)$$

$$b_{1,ES} = e_{Circ,Endo} [b_{1,ED}] \quad (3)$$

$$[b_{1,ES}^2 + a_{1,ES}^2] = e_{Long,Endo}^2 [b_{1,ED}^2 + a_{1,ED}^2] \quad (4)$$

where a_1 is the endocardial major semiaxis, a_2 is the epicardial major semiaxis, b_1 is the endocardial minor semiaxis, b_2 is the epicardial minor semiaxis, ED is end-diastole, ES is end-systole, and $e_{Circ,Endo}$ and $e_{Long,Endo}$ are endocardial circumferential and longitudinal strains, respectively. Equation 1 forces LV wall volume in diastole and systole to be equal. Equation 2 constrains the ventricular aspect ratio (ratio of wall thickness at the apex to the base) in diastole and systole to be equal. Equations 3 and 4 decrease the circumferential and longitudinal circumferences by $e_{Circ,Endo}$ and $e_{Long,Endo}$, respectively. $e_{Circ,Endo}$ and $e_{Long,Endo}$ were respectively chosen so that the average circumferential and longitudinal strains are equal to 0.85 (strain of an unloaded sarcomere during contraction).¹⁹⁻²¹

Finite element mesh and boundary conditions. The finite element model was meshed with axisymmetric solid (continuum) elements (4 nodes, bilinear with reduced integration, element type CAX4RH, ABAQUS, standard module, version 5.4, Hibbet, Karlson and Sorenson, Inc, Providence, RI). A single axisymmetric hydrostatic fluid-filled cavity (element type FAX2, ABAQUS) was used to model the endocardial cavity. This element provides a convenient means by which endocardial cavity pressure and volume can be calculated. Nodes at the apex along the ordinate axis were constrained to displace in the y direction (long axis) only. The base of the LV is composed primarily of fibrous tissue and was therefore constrained in all degrees of freedom (Fig 1, step 2).

Material properties.

Diastolic material properties. Both diastolic and systolic material properties of the LV wall were assumed to be homogeneous and isotropic. Diastolic material properties were described by the strain energy potential constitutive equation

developed by Ogden²² to describe nonlinear isotropic rubber-like materials. The Ogden constitutive equation is shown below:

$$U = \sum_{i=1}^N \frac{2\mu_i}{\alpha_i^2} [\lambda_1^{-\alpha_i} + \lambda_2^{-\alpha_i} + \lambda_3^{-\alpha_i} - 3] \quad (5)$$

where the strain energy, U , is the stored internal energy per unit volume, and extension λ_i is the length, l_i , divided by the undeformed reference length at zero force, l_{oi} . The parameters μ_i and α_i are material constants, α_i being termed the power law exponent. Determination of μ_i and α_i for the myocardium in different species, and by different methods, has yielded similar values.¹³ Stiffness parameters from the normal canine ventricle²³ where $\mu_1 = 16$ and $\alpha_1 = 16.0$ were used in the diastolic model.

Systolic material properties. The end-systolic material properties of the LV wall were also described by means of the Ogden constitutive equation. End-systolic material stiffness parameters were empirically chosen so that the end-systolic pressure-volume relationship is approximately linear and passes through LV pressure of 120 mm Hg and LV volume of 200 mL. In general, μ_i determines the initial slope of the pressure-volume relationship and α_i determines the shape of the relationship. Stiffness parameters of $\mu_i = 59.722$ and $\alpha_i = 9.5$ were empirically found to produce a nearly linear pressure-volume relationship and to pass through target LV pressure and volume. These stiffness parameters are significantly less stiff than those of normal systolic myocardium.¹³

Finally, both zero-pressure systole and zero-pressure diastole occur at $\lambda = 1$. However, the rest length is different for systole and diastole (equations 3 and 4).

VVRS simulations. The effects of VVRS were simulated with the use of the globally dilated heart model. Apical and lateral VVRS were tested at 80% and 90% of original ventricular mass. More specifically, 10% and 20% of LV mass was removed from both the unloaded diastolic and systolic shapes. Apical resections were performed by first determining the position of a simulated apical ventriculectomy (Fig 1, step 3, exploded view: dashed line) associated with a 10% or 20% LV mass reduction. The cut edge of the ventriculectomy was parallel to y (long) axis and had y-coordinates of $a_{1,AR}$ and $a_{2,AR}$. The cut edge of the ventricle was then moved to the midline ($x = 0$) (Fig 1, step 3, expanded view of apex: x) and the y-coordinates of the cut edge then became the new major semiaxes. Minor semiaxes were unchanged (Fig 1, step 3: b_1 and b_2).

Lateral resections were performed by first determining the position of a simulated lateral ventriculectomy (Fig 1, step 4) associated with a 10% or 20% LV mass reduction. The ventricle was incised parallel to the y (long) axis and the cut edges of the ventricle were apposed, creating new minor semiaxes (Fig 1, step 4: $b_{1,LR}$ and $b_{2,LR}$). The wall thickness was unchanged ($b_2 - b_1 = b_{2,LR} - b_{1,LR}$). Minor semiaxes were unchanged (Fig 1, step 4: a_1 and a_2). No attempt was made to alter material properties around the ventricular incision.

Calculation of systolic and diastolic pressure-volume relationships. Diastolic solutions were obtained at a range of

diastolic intracavitary pressures (ie, 0-40 mm Hg). End-systolic solutions were obtained at a range of systolic intracavitary pressures (ie, 0-120 mm Hg). The pressure, P_{ES} , and volume at end-systole, V_{ES} , derived from the finite element model were fit to the following linear equation by means of least squares regression analysis²⁴ (Microsoft EXCEL, Redmond, Wash):

$$P_{ES} = E_{ES} [V_{ES} - V_0] \quad (6)$$

where V_0 is the volume intercept and E_{ES} is the slope of the LV elastance. The pressure, P_{ED} , and volume at end-diastole, V_{ED} , were fit to the following quadratic equation with the use of least squares regression analysis²⁴ (Microsoft EXCEL, Redmond, Wash):

$$P_{ED} = \beta_0 + \beta_1 V_{ED} + \beta_2 V_{ED}^2 \quad (7)$$

where β_0 , β_1 , and β_2 are the stiffness parameters of the LV diastolic compliance.

Calculation of stroke work/ P_{ED} (Starling) and stroke work/ V_{ED} (PRSW) relationships. For each simulation (DCM, 10% and 20% lateral and apical VVRS), stroke work/ P_{ED} and stroke work/ V_{ED} relationships were calculated from the diastolic and systolic pressure-volume regressions, assuming that arterial elastance, E_A ,²⁵ was constant. E_A was calculated according to the following equation:

$$SV = \frac{V_{ED} - V_0}{1 + E_A/E_{ES}} \quad (8)$$

where SV is the stroke volume, V_0 is the volume intercept, and E_{ES} is the slope of the LV end-systolic elastance (equation 6). The initial LV pressure-volume loop ($ABCD$, Fig 2) was constructed so that $P_{ES} = 100$ and $P_{ED} = 20$. Initial E_A was calculated, P_{ED} was incrementally reduced, successive values of stroke volume were calculated with the use of equation 8, and corresponding values of V_{ES} and P_{ES} were used to construct successive pressure-volume loops. The stroke work/ P_{ED} and stroke work/ V_{ED} relationships were fit with a linear least squares regression²⁴ (Microsoft EXCEL, Redmond, Wash).

Calculation of pressure-volume area. For each simulation (DCM and 10% and 20% lateral and apical VVRS), pressure-volume area²⁶ was calculated from the initial pressure-volume loop ($P_{ES} = 100$ and $P_{ED} = 20$) and the diastolic and systolic pressure-volume regressions. Pressure-volume area is equal to the stroke work (area $ABCD$; Fig 2) plus the potential energy (area OCD , Fig 2). Pressure-volume area/mass is our attempt to normalize energy expenditure for different amounts of remaining LV myocardium. Pressure-volume area/mass is an average term and therefore an approximation given that different myocardial regions experience different stresses and therefore have different regional energy expenditures.

Results

End-systolic (A) and diastolic (B) finite element meshes of the globally dilated, poorly contractile LV before

Fig 2. Schematic illustration of cardiac function parameters. E_A , Arterial elastance; E_{ES} , end-systolic elastance; SW , stroke work (area $ABCD$); PVA , pressure-volume area (area $ABCOD$).

and after loading are seen in Fig 3. In each panel the unloaded mesh is in green and the loaded mesh is in red. In this and subsequent examples, only the right half of the axisymmetric mesh is shown, the diastolic model is loaded with 20 mm Hg of intracavitary LV pressure, and the systolic model is loaded with 100 mm Hg of intracavitary LV pressure. End-systolic (A) and diastolic (B) finite element meshes of 20% apical VVRS before and after loading are seen in Fig 4. End-systolic (A) and diastolic (B) finite element meshes of 20% lateral VVRS before and after loading are seen in Fig 5. Unloaded and loaded LV dimensions are seen in Table I. Notice the realistic end-systolic and end-diastolic dimensions of the model of dilated cardiomyopathy. The percent wall thickness change was 26% at the equator.

The effect of apical VVRS (apical resection, AR) on LV elastance and compliance is seen in Fig 6. Elastance curves are to the left and compliance curves are to the right. Ten-percent apical VVRS and 20% apical VVRS shift preresection LV elastance and compliance progressively to the left. In each case, the compliance is shifted a greater relative amount to the left than the elastance. The effect of lateral VVRS (lateral resection, LR) on LV elastance and compliance is seen in Fig 7. Elastance curves are to the left and compliance curves are to the right. Ten-percent lateral VVRS and 20% lateral VVRS shift preresection LV elastance and compliance progressively to the left. In each case, the compliance is shifted further to the left than the elastance. Also, for a given amount of resected LV mass, lateral VVRS (Fig 7) shifts compliance and elastance further to the left than apical VVRS (Fig 6).

The effect of VVRS on diastolic and end-systolic LV stiffness parameters is seen in Table II. Note that VVRS altered end-systolic function by increasing elastance from 0.826 to 1.83 and decreasing V_0 from 59 to 28 mL. VVRS altered diastolic function by increasing the

Fig 3. Globally dilated, poorly contractile LV model. Systolic (A) and diastolic (B) finite element mesh before and after loading. In each panel the unloaded mesh is in green and the loaded mesh is in red. In each case only the right half of the axisymmetric mesh is shown. The diastolic model is loaded with 20 mm Hg of intracavitary LV pressure. The systolic model is loaded with 100 mm Hg of intracavitary LV pressure.

coefficient of the V_{ED}^2 term, β_2 , from 0.002 to 0.005. In each case 20% lateral VVRS caused the maximum effect. Lateral VVRS had a larger effect on diastolic function than did apical VVRS.

The effect of VVRS on the stroke work/end-diastolic volume (PRSW) relationship is seen in Fig 8, A. Ten-percent apical VVRS, 20% apical VVRS, 10% lateral VVRS, and 20% lateral VVRS shift prerection PRSW relationship progressively to the left. Note that VVRS not only shifts the V_{ED} intercept to the left but increases the slope of the relationship as well (DCM 0.006 J/mL; 20% lateral VVRS 0.009 J/mL). Note that this effect is increased with a larger amount of resected LV mass and is greater in lateral VVRS than apical VVRS.

The effect of VVRS on the stroke work/end-diastolic pressure relationship (Starling) is seen in Fig 8, B. Ten-percent apical VVRS, 20% apical VVRS, 10% lateral VVRS, and 20% lateral VVRS progressively shift the end-diastolic pressure intercept to the left. Note that all curves intersect at approximately $P_{ED} = 5$ mm Hg. To the right of the point of intersection, VVRS causes a

progressive decrease in the Starling relationship slope (DCM 0.028 J/mm Hg; 20% lateral VVRS 0.023 J/mm Hg). However, the decrease is relatively small, causing only a 3-mm Hg difference between DCM and VVRS at stroke work of 0.5 J. Note that this effect is increased with a larger amount of resected LV mass and is greater in lateral VVRS than in apical VVRS. In summary, any amount of either apical or lateral VVRS decreases the slope of the Starling relationship but increases the slope of the stroke work/end-diastolic volume relationship.

The effect of VVRS on end-diastolic and end-systolic LV volume, stroke volume, and ejection fraction is seen in Table III. End-diastolic volume was obtained at 20 mm Hg and end-systolic volume was obtained at 100 mm Hg. Note that stroke volume decreases while ejection fraction increases with VVRS. Stroke volume was reduced in all cases because the decrease in diastolic compliance was not sufficiently compensated by the improvements in end-systolic elastance.

The effect of VVRS on the energy expenditure per gram of myocardium is seen in Fig 9. Note that apical VVRS

Fig 4. Apical VVRS: Systolic (A) and diastolic (B) finite element mesh before and after loading. In each panel the unloaded mesh is on the *left* and the loaded mesh is on the *right*. In each case only the right half of the axisymmetric mesh is shown. The diastolic model is loaded with 20 mm Hg of intracavitary LV pressure. The systolic model is loaded with 100 mm Hg of intracavitary LV pressure.

has an insignificant effect (1%) on myocardial energy expenditure. Ten-percent and 20% lateral VVRS reduces energy expenditure by 7% and 17%, respectively.

Discussion

The principal result of this finite element simulation of VVRS is that both apical and lateral VVRS shift diastolic compliance and end-systolic elastance to the left on the pressure-volume diagram. The net effect of VVRS on ventricular function is mixed. Any amount of either apical or lateral VVRS increases the slope of the stroke work/end-diastolic volume (PRSW) relationship. On the other hand, any amount of either apical or lateral VVRS decreases the slope of the Starling relationship. However, the decrease in the Starling relationship is small (3-mm Hg difference between DCM and VVRS at a stroke work = 0.5 J), and the increase in end-diastolic pressure and corresponding increase in left atrial pressure needed to attain comparable stroke work may be effectively offset by the removal of mitral regurgitation.

VVRS causes an increase in ejection fraction while stroke volume decreases. However, ejection fraction is a poor index of ventricular function,^{27,28} and it is therefore incorrect to conclude that cardiac function has improved if VVRS increases ejection fraction.

The effects of apical and lateral VVRS on compliance, elastance, and ventricular function are qualitatively similar. The effect of 20% lateral VVRS is quantitatively largest, whereas 10% lateral VVRS is similar to 20% apical VVRS. At present, cardiac surgeons resect either an ellipse of lateral free wall myocardium or an inverted triangle or "shield," in which the wide part of the triangle or shield is toward the atrioventricular groove. However, an apical resection would be easier to surgically reconstruct and might not interfere with the papillary muscles. These results support the potential use of apical partial ventriculectomy.

VVRS causes a decrease in energy expenditure per gram of residual myocardium. Although not calculated in this study, wall stress throughout systole is also decreased.^{6,27} Both of these factors may lead to a

Fig 5. Lateral VVRS: Systolic (A) and diastolic (B) finite element mesh before and after loading. In each panel the unloaded mesh is on the left and the loaded mesh is on the right. In each case only the right half of the axisymmetric mesh is shown. The diastolic model is loaded with 20 mm Hg of intracavitary LV pressure. The systolic model is loaded with 100 mm Hg of intracavitary LV pressure.

reverse remodeling of eccentric hypertrophy and may lead to potential subacute and chronic beneficial effects from VVRS. The overall effect of VVRS must be a combination of the acute hemodynamic effects of VVRS on LV function and subacute and chronic effects of decreased energy expenditure and stress on ventricular hypertrophy.

Finite element modeling and assumptions. The accuracy of finite element analysis simulations are dependent on the accuracy of initial assumptions, including (1) the material property law, (2) the input stiffness parameters, and (3) the initial unloaded structural geometry. Given accurate initial conditions, however, finite element analysis is well suited to model the effect of LV volume reduction surgery on elastance, compliance, and ventricular function. However, the following assumptions must be recognized.

Structure. Although more realistic than previous models, the initial end-diastolic and end-systolic shapes are approximate. This is in part because there are no experimental measurements of the normal or

pathologic animal or human LV at zero left and right ventricular intracavitary pressure. Furthermore, Fung²⁹ has demonstrated significant residual wall stress even in the absence of ventricular pressure. Residual stress has not been measured in dilated cardiomyopathy but may have a significant impact on VVRS, which in effect increases residual pre-strain and stress.

The right ventricle is not included in the model, and transeptal pressure is assumed to be the same as transmural pressure in the lateral LV wall. Right ventricular pressure has been shown to increase LV elastance and compliance,³⁰ and the absence of the right ventricle may lead to a shift in elastance and compliance to the right.

Although finite element techniques exist that can model interaction between fluid motion in a chamber and the structure and stiffness of the chamber wall, our model does not have that capability and does not incorporate the effect of mitral regurgitation. This may be of particular importance in the interpretation of the stroke work/end-diastolic pressure curves (Fig 8, A). As

Fig 6. Elastance and compliance before and after apical VVRS. Elastance curves are to the *left* and compliance curves are to the *right*. DCM, Dilated cardiomyopathy; AR, apical VVRS. Ten-percent AR (*open triangles*) and 20% AR (*open circles*) progressively shift preresection LV elastance and compliance (*asterisks*) to the left. In each case note that the compliance is shifted further to the left than the elastance. Symbols (*asterisk*, *open triangle*, and *open circle*) represent the actual values calculated by the finite element solver.

above, the increase in end-diastolic pressure and corresponding increase in left atrial pressure needed to attain comparable stroke work may be effectively offset by the removal of mitral regurgitation.

The model was constructed so that average circumferential and longitudinal unloaded strains across the LV wall were equal to 0.85. This, in turn, is based on the relative change in sarcomere length between the relaxed and the contracted states. Spotnitz, Sonnenblick, and Spiro¹⁹ measured sarcomere length in normal intact hearts and found sarcomere lengths between 1.87 and 1.95 μm in diastole and between 1.5 and 1.6 μm in systole. These values agree with values from isolated intact and skinned ventricular muscle strips.^{20,21}

Material properties. Scientists have used different types of material property laws in finite element analysis models. Material property laws have ranged from Hookean (linear stress-strain relation)³¹ to nonlinear,¹³ and both isotropic¹³ and anisotropic (directional)³² descriptions have been used. In addition, although an anisotropic material property law that is linked to myocardial architecture (fiber angles) is clearly preferable,³²⁻³⁴ these descriptions are complex and knowledge of myocardial fiber angles in the dilated cardiomyopathic LV is unknown. A nonlinear isotropic material property law, such as that developed by Ogden²² to describe nonlinear isotropic rubber-like materials, represents a reasonable first-order approximation until such structural information becomes available.

Fig 7. Elastance and compliance before and after lateral VVRS. Elastance curves are to the *left* and compliance curves are to the *right*. DCM, Dilated cardiomyopathy; LR, lateral VVRS. Ten-percent LR (*closed triangles*) and 20% LR (*closed circles*) progressively shift pre-resection LV elastance and compliance (*asterisks*) to the left. In each case note that the compliance is shifted further to the left than the elastance. Note that for each amount of resected LV mass, lateral VVRS shifts compliance and elastance further to the left than apical VVRS (Fig 5). Symbols (*asterisk*, *closed triangle*, and *closed circle*) represent the actual values calculated by the finite element solver.

Although the incision probably undergoes stiffness changes similar to other wounds and myocardial infarction,³⁵ stiffness in the region of the incision has not been experimentally measured. Therefore no attempt was made to alter material properties around the ventricular incision. Also, the fibrous base of the LV is assumed to be fixed in the diastolic position.

Finite element mesh. The finite elements used are 2-dimensional axisymmetric elements. Although these elements are computationally efficient, they can only be used to describe axisymmetric solids in rotation and cannot be used to describe variation in the third dimension.

VVRS in the apical position was performed by “gathering” the LV at the point of resection. True resection with linear closure in the operating room would lead to a more complex non-axisymmetric 3-dimensional shape. As above, no attempt was made to alter material properties around the ventricular incision.

Mathematical models of VVRS. There are analytic solutions that relate chamber compliance to stress and strain in simple shapes with linear material properties that undergo small deformations. An example is the linear elastic solution of an incompressible thin-walled sphere under internal pressure described by Lamé,³⁶

$$\frac{\partial P}{\partial V} = \frac{Et}{\pi a^4} \quad (9)$$

Table I. Dimensions of unloaded and loaded LV

Model	Unloaded model dimensions				Loaded model dimensions			
	a1 (cm)	b1 (cm)	a2 (cm)	b2 (cm)	a1 (cm)	b1 (cm)	a2 (cm)	b2 (cm)
Systolic								
DCM	9.0	1.7	9.7	3.6	11.4	1.7	11.9	3.6
10% AR	7.8	1.7	8.7	3.6	10.0	1.7	10.5	3.6
20% AR	6.3	1.7	7.5	3.6	8.0	1.7	8.9	3.6
10% LR	9.0	1.4	9.7	3.4	11.2	1.4	11.6	3.4
20% LR	9.0	1.2	9.7	3.1	10.9	1.2	11.3	3.1
Diastolic								
DCM	10.7	2.3	11.2	3.8	12.7	2.3	13	3.8
10% AR	9.4	2.3	10.0	3.8	11.1	2.3	11.6	3.8
20% AR	7.8	2.3	8.7	3.8	9.2	2.3	9.9	3.8
10% LR	10.7	2.0	11.2	3.5	12.6	2.0	12.9	3.5
20% LR	10.7	1.7	11.2	3.2	12.4	1.7	12.8	3.2

DCM, Dilated cardiomyopathy; AR, apical resection (apical VVRS); LR, lateral resection (lateral VVRS); a1, endocardial long axis; a2, epicardial long axis; b1, endocardial short axis; b2, epicardial short axis radius (Fig 1, step 2). The relationship between unloaded diastolic and end-systolic dimensions was defined by equations 1 to 4, which were solved simultaneously. Loaded diastolic dimensions were obtained at 20 mm Hg and loaded end-systolic dimensions were obtained at 100 mm Hg.

Table II. The effect of VVRS on diastolic compliance, end-systolic elastance, stroke work/end-diastolic volume, and stroke work/end-diastolic pressure (Starling) relationships

Model	End-systolic elastance		Diastolic compliance			SW/ V_{ED}	SE/ P_{ED}
	E_{ES}	V_0	β_0	β_1	β_2	m_{VED}	m_{PED}
DCM	0.83	58.96	33.7	-0.445	0.002	0.006	0.028
10% AR	0.97	50.83	29.0	-0.466	0.002	0.006	0.024
20% AR	1.25	40.40	35.6	-0.655	0.003	0.007	0.023
10% LR	1.18	41.53	27.0	-0.504	0.003	0.007	0.025
20% LR	1.83	27.54	25.6	-0.668	0.005	0.009	0.023

DCM, Dilated cardiomyopathy; AR, apical resection (apical VVRS); LR, lateral resection (lateral VVRS); SW, stroke work; V_{ED} , end-diastolic volume; P_{ED} , end-diastolic pressure. V_0 , volume intercept; E_{ES} , slope of the LV elastance. β_0 , β_1 , β_2 , stiffness coefficients of the quadratic function (equation 7) used to model LV diastolic compliance. m_{VED} , m_{PED} , slopes of the stroke work/end-diastolic volume and Starling relationships, respectively.

Table III. The effect of VVRS on end-diastolic and end-systolic LV volume, stroke volume, and ejection fraction

Model	ED volume (mL)	ES volume (mL)	Stroke volume (mL)	Ejection fraction (%)
DCM	242.8	180.1	62.8	25.8
10% AR	211.8	154.4	57.4	27.1
20% AR	176.8	120.3	56.5	32.0
10% LR	186.6	126.7	59.9	32.1
20% LR	136.2	82.4	53.8	39.5

DCM, Dilated cardiomyopathy; AR, apical resection (apical VVRS); LR, lateral resection (lateral VVRS). Diastolic volume was obtained at 20 mm Hg and end-systolic volume was obtained at 100 mm Hg. Note that stroke volume decreases while ejection fraction increases.

where a and t are, respectively, the internal radius and the wall thickness of the sphere and E is the modulus of elasticity. Furthermore, the thin-walled sphere solution is qualitatively consistent with the results of our finite

element model in that both predict an increase in chamber stiffness if internal radius is decreased and wall thickness is maintained. Nevertheless, our objective in developing a large deformation ellipsoidal finite element model of partial ventriculectomy is to improve the *quantitative* aspects of this type of analysis. There are no analytic solutions for a thick-walled ellipse. The finite element method is the only way to incorporate realistic (although simplified) ventricular geometry and measured material properties in a structure that undergoes large deformations.

Despite its shortcomings, our model in conjunction with diastolic constitutive parameters derived from actual resting stress-extension tests of canine myocardium produces realistic human pressure-volume curves. Proceeding in the other direction (ie, starting with a time varying elastance model and then characterizing stress-strain behavior by way of global wall equilibrium) could very well produce the correct trend while giving quantitatively unreliable data.

Fig 8. The effect of VVRS on the stroke work/end-diastolic volume (A) and stroke work/end-diastolic pressure (B) (Starling's law) relationship. Ten-percent AR (dash-dot), 20% AR (dot-dot), 10% LR (dash-dot-dot), and 20% LR VVRS (dash-dot-dot-dot) progressively shift stroke work/end-diastolic volume relationship to the left (A). Note that VVRS not only shifts the V_{ED} intercept to the left but increases the slope of the relationship as well (DCM 0.006 J/mL; 20% lateral VVRS 0.009 J/mL). On the other hand, VVRS progressively decreases the slope of LV Starling relationship (B) above end-diastolic pressure = 5 mm Hg. Note that these effects are increased with a larger amount of resected LV mass and are greater in lateral VVRS than apical VVRS.

Comparison with preliminary clinical results. Early reports have documented a heterogeneous effect of VVRS on ventricular function.^{3,5} End-diastolic and end-systolic LV volumes have been consistently decreased^{4,10} and ejection fraction has consistently increased.^{4,5,7} Although most reports document a decrease in stroke volume,^{4,10} stroke volume⁸ and cardiac index⁵ have been noted to improve. However, because postoperative ventricular end-diastolic pressure is often decreased^{4,5} and heart rate increased,⁴ these data are difficult to interpret.

Fig 9. The effect of VVRS on the energy consumption per gram of myocardium. Note that 10% LV mass resection has an insignificant effect (1%) on myocardial energy expenditure. The 20% apical VVRS and 20% lateral VVRS reduce energy expenditure by 7% and 17%, respectively. PVA, Pressure-volume area.

The effects of VVRS on load-independent end-systolic and diastolic pressure-volume relationships have been measured with ventriculography,⁹ conductance catheters,¹⁰ and 2-dimensional echocardiography.³ End-systolic elastance has been found to increase by twofold to threefold^{9,10} and end-diastolic compliance significantly decreases.^{3,10} Because of the heterogeneous effect of VVRS on end-systolic elastance in their study, Gorscan and associates³ found that PRSW was not changed. The Starling relationship has not been measured. Although these preliminary results are consistent with our finite element analysis model and support the findings of this study, the effect of VVRS on ventricular function (Starling and PRSW) needs to be studied in more detail.

Conclusion and future directions

Batista's operation is an intriguing and potentially important surgical therapy for patients with DCM and congestive failure. However, although seemingly intuitive, the effect of partial ventriculectomy on ventricular function is complex. We suggest that this and all operations that surgically remodel ventricular size, shape, or regional stiffness should be analyzed by determining how the procedure affects both systolic and diastolic pressure-volume relationships and how those changes affect ventricular function.

Our finite element simulation of Batista's operation documents an acute increase in the stroke volume/end-diastolic volume relationship. However, decreased diastolic compliance causes a small decrease in the Starling relationship (3 mm Hg difference between DCM and VVRS at stroke work = 0.5 J). On the other

hand, the increase in left atrial pressure needed to maintain stroke work may be effectively offset by the surgical correction of mitral regurgitation. The overall effect of VVRS is a combination of the acute hemodynamic effects of VVRS on LV function and subacute and chronic effects of decreased energy expenditure and stress on ventricular hypertrophy. This analysis suggests that VVRS may be an appropriate therapy for patients with DCM.

This model is an initial approximation. However, one of the major strengths of the finite element method is the ability to incorporate complex realistic geometry, anisotropic material properties, regional differences in material properties (ie, at the suture line), and residual stress in the unpressurized ventricle. In the future, we intend to progressively refine the model. Simulations should be repeated with varied systolic and diastolic material parameters. In addition, future directions should include the measurement of unloaded LV systolic and diastolic shapes in both the normal and pathologic heart. However, we believe that this initial approximation provides useful information provided that the assumptions incorporated in the model are clearly understood.

The finite element approach can be used to analyze the effects of any cardiac operation that surgically remodels ventricular size or shape or changes regional stiffness. Ideally, the development of such simulations will refine our understanding of operations and pathologic processes that alter ventricular shape and material properties.

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