A FINITE ELEMENT MODEL OF THE HUMAN VENTRICLES DURING THE RAPID FILLING PHASE

D.-E. Bettendorff-Bakman*, P. Schmid*, P.P. Lunkenheimer** and P. Niederer*

* Institute for Biomedical Engineering, University of Zürich and Swiss Federal Institute of Technology, Zürich, Switzerland

** Experimental Thoraco-vascular and Cardiac Surgery, University of Münster, Münster, Germany

bakman@biomed.ee.ethz.ch

Abstract: The diastolic phase of the cardiac cycle is constituted by the isovolumetric relaxation and the blood filling of the ventricles. At the beginning of the filling process, the blood is immediately sucked into the ventricles in a short period called the rapid filling phase. During this period more than half of the subsequent stroke volume is entering the ventricles of the human heart quickly and the ventricular volumes double approximately. During the rapid filling phase, the ventricular pressures rise only by an amount of typically less than 1 kPa, that cannot be the sole driving mechanism of the large inflow of blood during this period. To investigate the role of the diastolic ventricular suction in this intensive volume augmentation, an elastic three-dimensional Finite Element model of both human ventricles has been developed, taking into account the effective compressibility of the myocardium. The end-sytolic geometry was obtained from Magnetic Resonance images. The results were compared with those obtained with a simpler Finite Element ellipsoid-model of the left cavity, as well as with experimental measurements of the human left ventricular volume. We conclude from this study that under normal conditions the ventricular suction plays an important role in the ventricular filling.

Introduction

The analysis of the stress-strain pattern of the myocardium during the cardiac cycle is aimed at elucidating the mechanics of the heart under physiologic and pathologic conditions. The variation in time of the intravascular fluid flow during one single heartbeat induces an effective compressibility of the otherwise incompressible cardiac tissue. Furthermore the ventricles are thick-walled and submitted to large deformations during the cardiac cycle.

The heart cycle is constituted by the ventricular contraction phase or systole and the diastolic phase, where the left (LV) and right (RV) ventricles are filled with blood while the myocardium is in a relaxed state. During ventricular systole, large amounts of blood accumulate in the atria because of the closed atrioventricular valves. The filling phase sets in immediately after the isovolumetric relaxation period, at the end of which the

ventricular pressures approach their diastolic values. The tricuspid and mitral valves open because the pressures in the atria are moderately higher than the intraventricular pressures due to the venous return. As the myocardium returns from the contracted to its resting state, the blood flows rapidly from the atria into the ventricles and their volumes increase quickly. This is denoted as the rapid filling phase and represents about the first third of diastole. During the middle third only a small amount of blood flows into the ventricles, corresponding to the momentary venous return reaching the ventricles through the atria. The rise of the ventricular volumes is small. Finally the atria contract and give an additional thrust to the ventricular blood filling. This period is called the atrial systole. It corresponds to about 25 percent of the filling phase.

From a mechanical point of view, the filling process of the ventricles has long been seen as a passive mechanism, depending principally on the venous return and atrial contraction. Submitted to the diastolic pressures, the relaxed ventricles are filled with blood and the myocardial tissue of the ventricular wall is stretched. Given that blood flowing into the left cavity is first pumped through the lungs by the right ventricle, the cardiac output is, in this traditional representation of diastole, solely monitored by the venous filling pressure.

In view of the low value of the filling pressure (less than 1 kPa) and considering that the ventricles of the human heart double in volume during the first third of diastole at rest, this classical view of ventricular filling has been questioned. On the basis of experiments with isolated perfused or excised beating intact hearts of mammalians [1, 2, 3, 4], a model for the ventricular rapid filling phase has been proposed [5]. It is founded on a mechanism driven by energy release during early diastole: During the systolic contraction of the myocardium, a part of the increasing potential energy of the heart, due to the compression of its muscle fibers and elastic elements, is stored in the elastic components of the cardiac muscle. At the beginning of diastole, this stored energy is released through the deformation of the heart and serves to displace blood into the ventricles when the mitral and tricuspid valves open. Contracting, during systole, below their equilibrium volume, both ventricles are inclined naturally to expand even without blood filling at the start of the diastolic phase, as a stretched elastic spring tends to return to its resting length when the stretch force is removed. This ventricular expansion, caused by restoring forces that act in the myocardial wall, generates a negative pressure and the blood is immediately sucked from the atria into the ventricles during the rapid filling phase. Recent studies on canine [6] and human [7] left cavities indicate that the systolic twisting of the ventricles, with torsion at the base and apex being of opposite sense, can also be an important mechanism for the heart to store potential energy during systole. This energy can then be liberated at the beginning of the diastolic phase through the ventricular untwisting, providing internal restoring forces that are responsible for the ventricular blood suction during early diastole. In this way, the rapid filling phase can be seen as the period where the heart recoils from its end-systolic configuration.

In order to study the influence of ventricular suction on the rapid filling phase, a three-dimensional elastic Finite Element (FE) model of both human ventricles has been developed, including a realistic geometry and taking into account the effective compressibility of the myocardial tissue.

Materials and Methods

End-systolic geometry

Based on earlier work [8, 9], a Finite Element model of the human left and right ventricles was derived whose geometry was obtained from 32 short axis Magnetic Resonance (MR) slices extending from the apex to the base of a healthy human volunteer at end-systole. The left and right ventricular volumes at end-systole were 50 ml. These volumes lie at the upper limit of the physiological range, which extends typically from some 30 ml to about 50 ml. Furthermore, the end-systolic volume of the myocardial wall was around 92 ml, which likewise corresponds to a typical physiological value. The model consisted of 3837 parabolic 20-node hexahedral and 10-node tetrahedral elements, respectively, yielding some 57700 degrees of freedom (see Fig. 1).

In order to validate the results obtained with this model utilizing a realistic geometry, a simple threedimensional model of the left ventricle has also been developed. In this model, the geometry of the left cavity was approximated to a stretched thick-walled spheroid, whose internal volume was 50 ml. It contained 400 parabolic 20-nodes hexahedral elements, producing some 7000 degrees of freedom.

Quadratic interpolation functions were used for the representation of the coordinates and displacements. To obtain the stiffness of the hexahedral and tetrahedral element, 27-point and 4-point Gaussian integration was



Figure 1: End-systolic geometry of both ventricles of the human heart

used, respectively, in the Finite Element software MSC Marc Mentat.

Material Properties

In the presented work, the myocardium was considered as a homogeneous, isotropic material. The study was performed under the assumption of linear elasticity allowing for large deformations. The stress-strain relation is described by the Kirchhoff-St. Venant law:

$$S_{ij} = \lambda \cdot Tr(E) \cdot \delta_{ij} + 2 \cdot G \cdot E_{ij} \tag{1}$$

where S_{ij} are the ij components of the second Piola-Kirchhoff stress tensor *S*, associated to the Green-Lagrange strain tensor *E*. λ and *G* (elastic shear modulus) are the Lamé coefficients and are related to the Young modulus *C* and the Poisson's ratio *v*.

In this study, the Young modulus was set to 20 kPa [10] and the effective compressibility of the myocardium, due to the coronary blood and lymphatic flows, was simulated with a value of 0.4 for the Poisson's ratio.

Boundary and initial conditions

Measurements of the evolution of the healthy human left ventricular volumes [11, 12, 13] at rest during the rapid filling phase were adopted as target values for the present FE analyses.

In all our simulations, a linear elastic foundation with a stiffness k_f was applied on the epicardium to simulate the influence of the surrounding tissue on the heart. Values of k_f were estimated by making parametric FE analyses with the boundary conditions described below. In this study, k_f was varied from 0.02 kPa to 0.1 kPa. Furthermore, for all nodes at the base, the degree of freedom in the long axis direction was suppressed and the nodes of the apex of the left ventricle were fixed laterally.



Figure 2: Procedure to calculate a single step in the FE simulation of ventricular suction in the rapid filling phase. A preliminary simulation derives the stress repartition in the myocardial wall from the initial geometry and the ventricular pressures. The inverse stress repartition is then applied to the initial geometry to obtain the deformed geometry under the influence of ventricular suction.

To simulate the ventricular suction in the rapid filling phase, it was necessary to determine the restoring forces acting in the myocardium during this process. For this purpose, preliminary FE simulations were performed, where both ventricles were inflated with linear time dependent pressure functions. As it has been shown in [11], a total pressure of more than 2 kPa was necessary in order to obtain the measured rise [13] in the left ventricular volume during the rapid filling phase. Therefore, the internal pressure applied on the endocardium of the left cavity, in both FE models, increased from 0 kPa to 2.3 kPa during the first third of diastole. For the right ventricle, its pressure was assumed to be, in the FE model using the realistic geometry, 40 percent of that of the left ventricle, corresponding to a pressure increase of 0.9 kPa in agreement with general physiology.

Inertial and gravitational forces were neglected. The loading was divided into 10 successive steps, except for the simulation, where k_f was set to 0.021 kPa. In this case, due to the low rigidity of the elastic foundation, unrealistic local deformations occur in the model of both human ventricles, causing convergence problems when the FE analysis was performed in too large steps. Therefore, for k_f equal to 0.021 kPa, the simulation of

the rapid filling phase with the FE model derived from Magnetic Resonance Imaging (MRI) measurements was done in 11 steps. For each load step the solution was calculated iteratively using the full Newton-Raphson method.

For the FE model of both human ventricles, the initial geometry of the first step (i = 1) corresponded to the end-systolic geometry G_0 , obtained from MRI measurements. For the simple FE model of the left cavity, it was represented by the ellipsoid with an internal volume of 50 ml. Fig. 2 shows the procedure used to calculate a single step i. For clarity reasons, only the base of both ventricles is displayed. First, a preliminary FE analysis was performed, where the initial geometry (G(i-1)) corresponded to the deformed geometry resulting from the preceding step i-1. The incremental linear time-dependent pressure functions $p_{LV}(i-1)$ and $p_{RV}(i-1)$ were applied, in this preliminary simulation, to the surfaces of the elements, representing the endocardium of the left and right ventricles, respectively (Fig. 2(1)). Under the influence of these ventricular pressures, the internal volumes of both cavities increase and the pressure-deformed geometry G(i), containing internal stresses $\overline{S(i)}$, is obtained (Fig. 2(2)).

The initial geometry (G(i-1)) from the preliminary simulation was subsequently taken as original geometry for the definite calculation of step i. The ventricular suction observed during the rapid filling phase was modeled by applying, as initial conditions, the internal stresses S(i) as calculated above to each integration point of the elements in reversed direction (Fig. 2(3)). In Figs. 2(2) and 2(3)), the distribution of the internal stresses in the model is visualized by the repartition of the equivalent Von Mises stress $S_{Eq.VonMises}$ in both ventricles. Under the action of these restoring forces, represented by the internal stresses S(i) (S(i) = -S(i)), both ventricles expand, yielding the deformed geometry G(i) (see Fig. 2(4)). In theory, this geometry G(i) is the same as the pressuredeformed geometry G(i) obtained in the above-described preliminary FE analysis. Then, G(i) was subsequently used as initial geometry for the step i+1.

Results

Fig. 3 (top) shows the initial geometries with internal stresses at the last step of the FE analysis (i = 11 and i= 10, respectively), for the MRI-derived model of both human ventricles and for the simpler ellipsoid model of the left cavity. Under the action of these restoring forces, the internal volumes of the left and right cavities increase from about 86 ml and 87 ml, respectively, to reach in both cases an internal volume of around 90 ml at the end of the first third of diastole (see Fig. 3 (center, left)). The same internal volume of the left cavity was obtained at the end of the rapid filling phase under the influence of ventricular suction, in the case of the stretched thick-walled spheroid used to model the left ventricle (see Fig. 3 (center, right)). Fig. 3 (bottom) displays the distribution of the total displacement, relatively to the undeformed systolic geometries, in the myocardial wall at the end of the rapid filling phase. Furthermore, the initial internal stress repartition, as well as the residual stress distribution, in both FE models are represented in Fig. 3 (top and center, respectively) by the equivalent Von Mises stress repartition in the ventricular wall. All these results are shown in the case where k_f was set to 0.021 kPa, for both geometries. Similar results were obtained for values of k_f ranging from 0.02 kPa to 0.023 kPa. Greater values of k_f than 0.023 kPa caused unrealistic local deformations in the model of both human ventricles, due to an unbalance between geometry, boundary conditions, initial conditions and mechanical properties.

Discussion

The FE model of both ventricles, using a realistic geometry, yields similar results for the left cavity to those obtained with the simpler ellipsoid FE model of the left ventricle (see Fig. 3), except for the distribution of the total displacement (Fig. 3 (bottom)). The difference in the displacement repartition in the left cavity of both

FE models during the rapid filling phase is due to the influence of the right cavity on the left ventricular deformation. Because of the absence of the right ventricle, the ellipsoid-model of the left cavity is free to move radially, except at the apex, where the displacement is minimal (ranging from 0 to 1 mm). So in this simple model, at the end of the first third of diastole, the displacement is maximal (about 7 mm) at the endocardium near the base, decreases through the ventricular wall as well as from the base to the apex (Fig. 3 (bottom, right)). In the FE model of both ventricles, the restoring forces in the myocardial wall, calculated by the inflation of both cavities with internal pressures in the preliminary FE analysis, push the right ventricular side of the septum slightly towards the left ventricular one (see Fig. 3 (top, left)), preventing the free radial expansion of the left cavity. Therefore in Fig. 3 (bottom, left), the septum moves moderately (displacement ranging from 0 to 2 mm) and the displacement is maximal (between 9 and 10 mm) near the base at the endocardium of the free wall of both ventricles, opposite to the septum. As in the ellipsoid-FE model of the left cavity, the displacement decreases from the endocardium to the epicardium and from the base to the apex.

As predicted by an analytic model of the left ventricle [14], the initial internal stress in both FE models, is maximal at the endocardium, decreases in the myocardium to reach a minimal value at the epicardium (see Fig. 3 (top)). Furthermore, the order of magnitude of the initial internal stresses in the left ventricle is the same for the MRI-derived FE model as for the simpler ellipsoid FE model. Because in the realistic geometry of the heart, the convexity of the cavities varies along the ventricular surfaces, there is local accumulation of maximal stresses near the points of inflexion, where the convexity changes and where the curvature is higher, especially in the right ventricle of the MRI-derived model (see Fig. 3 (top, left)).

Under the influence of ventricular suction, the internal volume of the left cavity, in both FE models with a soft elastic foundation ($k_f = 0.021$ kPa), increases from 50 ml (corresponding to the end-systolic LV volume) to 90 ml. This corresponds to the measured rise in the human left ventricular volume [13] during the rapid filling phase. A decisive determinant for physiological verisimilitude is that at rest, on the average, left ventricular and right ventricular stroke as well as filling volumes are the same. This implies that at the beginning as well as at the end of the rapid filling phase the left and right ventricular volumes are equal within a few percent under homeostatic conditions. With the material parameters chosen, this important condition is satisfied in our FE analysis of the first third of diastole, when the rigidity of the elastic foundation k_f was set to 0.021 kPa, yielding the same increase in volume of the right cavity as in the left. Furthermore, in this case, there is an augmentation of the myocardial



Figure 3: Results obtained with both FE models at the last step of the elastic FE simulation of the rapid filling phase with $k_f = 0.021$ kPa. Top: Initial internal stresses in the ventricular wall, representing the restoring forces acting in the heart. Centre: Deformed geometries at the end of the rapid filling phase with residual stresses. Bottom: Total displacement repartition in the myocardium at the end of the rapid filling phase. Here the stresses (top and center) are represented by the equivalent Von Mises stresses $S_{Eq.VonMises}$.

wall volume of about 5% in both FE models. This is in agreement with the physiological volume change of the ventricular wall during early diastole, due to the coronary blood and lymphatic flows. As it is shown in Fig. 3 (center), for both FE models, the left ventricle is stress-free at the end of the rapid filling phase. Some local residual stresses are present in the right cavity, but comparing to the repartition of the applied internal stresses (Fig. 3 (top, left)), they are only located in few local areas where the convexity changes are important (Fig. 3 (center, left)).

Conclusions

From the results, presented here, we can conclude that our FE analysis of the ventricular suction during early diastole produced a realistic filling of both cavities. This indicates that the concept of the heart recoiling from its end-systolic configuration is an important mechanism for the rapid filling phase of the ventricles. The role of ventricular suction may be enhanced during exercise, where myocardial contractility is increased and the end-systolic internal volumes of both cavities are smaller, helping to augment the filling of the ventricles in order to provide an important cardiac output at a rapid cardiac rhythm [15].

References

- [1] BLOOM, W. L. Diastolic filling of the beating excised heart. *Am. J. Physiol.*, 187:143–144, 1956.
- [2] BRECHER, G. A. Critical review of recent work on ventricular diastolic suction. *Circ. Res.*, 6:554–566, 1958.
- [3] HORI, M., YELLIN, E. L., and SONNENBLICK, E.
 H. Left ventricular diastolic suction as a mechanism of ventricular filling. *Jpn. Circ. J.*, 46:124–129, 1982.
- [4] TYBERG, J. V., KEON, W. J., SONNENBLICK, E. H., and URSCHEL, C. W. Mechanics of ventricular diastole. *Cardiovasc. Res.*, 4:423–428, 1970.
- [5] ROBINSON, T. F., FACTOR S. M., and SONNEN-BLICK, E. H. The heart as a suction pump. *Sci. Am.*, 254:62–69, 1986.
- [6] RADEMAKERS, F. E., BUCHALTER M. B., ROGERS, W. J., ZERHOUNI, E. A., WEISFELDT, M. L., WEISS, J. L., and SHAPIRO, E. P. Dissociation between left ventricular untwisting and filling. accentuation by catecholamines. *Circulation*, 85:1572–1581, 1992.
- [7] KERNBERG, M. E., DAUGHTERS, G. T., ALDER-MAN, E. L., and INGELS, N. B. JR. Measurement of three-dimensional systolic torsional deformation and diastolic recoil rates in the human left ventricle. *Circulation*, 62(suppl. III:III–204, 1980.
- [8] SCHMID, P., STUBER, M., BÖSIGER, P., HESS, O. M., and NIEDERER, P. Determination of displacement, stress- and strain-distribution in the human heart: a fe-model on the basis of mr imaging. *Technol. Health Care*, 3:209–214, 1995.
- [9] SCHMID, P., NIEDERER, P., LUNKENHEIMER, P. P., and TORRENT-GUASP, F. The anisotropic structure of the human left and right ventricles. *Technol. Health Care*, 5:29–43, 1997.
- [10] GHISTA, D. N., SANDLER, H., and VAYO, W. H. Elastic modulus of the human intact left ventricle-determination and physiological interpretation. *Med. Biol. Eng.*, 13:151–161, 1975.
- [11] BETTENDORFF-BAKMAN, D.-E., SCHMID, P., LUNKENHEIMER, P. P., and NIEDERER, P. A finite element study relating to the rapid filling phase of the human ventricles. *J. Theor. Biol.*, in press:available online, 2005.
- [12] HOFF, F. L., TURNER, D. A., WANG, J. Z., BAR-RON, J. T., CHUTUAPE, M. D., and LIEBSON, P. R. Semiautomatic evaluation of left ventricular di-

astolic function with cine magnetic resonance imaging. *Acad. Radiol.*, 1:237–242, 1994.

- [13] NONOGI, H., HESS, O. M., RITTER, M., and KRAYENBUEHL, H. P. Diastolic properties of the normal left ventricle during supine exercise. *Br*: *Heart J.*, 60:30–38, 1988.
- [14] MIRSKY, I. Effects of anisotropy and nonhomogeneity on left ventricular stresses in the intact heart. *Bull. Math. Biophys.*, 32:197–213, 1970.
- [15] UDELSON, J. E., BACHARACH, S. L., CANNON, R. O. III, and BONOW, R. O. Minimum left ventricular pressure during beta-adrenergic stimulation in human subjects. evidence for elastic recoil and diastolic suction in the normal heart. *Circulation*, 82:1174–1182, 1990.