Macroscopic three-dimensional motion patterns of the left ventricle

CHAPTER 37

MACROSCOPIC THREE-DIMENSIONAL MOTION PATTERNS OF THE LEFT VENTRICLE

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ABSTRACT

The pattern of displacements in the left ventricle (LV) can be described by 13 modes of motion and deformation. Three functional modes of deformation are essential for ejection: a decrease in cavity volume, torsion, and ellipticalization. Four additional modes are used to describe asymmetric deformation. Six modes of rigid body motion describe rotation and translation. In the LV 14–20 radiopaque markers were inserted in the wall of the LV. They were distributed more or less evenly from base to apex and around the circumference. Torsion and volume changes require the definition of a cardiac coordinate system. The point at which ejection focusses is used as the origin, and the torsion axis is used as the z-axis. In the present study the coordinate system was positioned objectively by a least squares fit of the kinematic model to the measured motion of markers. In five dogs in the control state the kinematic parameters were determined as a function of time for all 13 modes. The torsion axis was displaced 4 ± 2 mm (mean±sd) from the center of the cross-section of the LV towards the lateral free wall. The direction of the torsion axis closely coincided with anatomical landmarks at the apex and base. During systole, a unique relation was found between the ratio of cavity volume to wall volume and torsion. This relation was universal to all LVs, the cylinder-symmetric mathematical model of cardiac mechanics inclusive. In diastole the patterns of deformation seem less universal and reproducible.

INTRODUCTION

The complicated motion pattern of the left ventricle (LV) may be simplified considerably by describing it as a sequence of appropriately chosen major modes of

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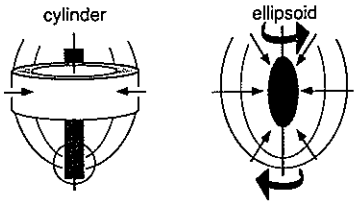

Figure 1. Left: In a cylindrical model, the ejected (shaded) region crosses the LV wall at the apex. **Right:** Using an ellipsoidal model for ejection, the ejected region is separated from the wall. The functional modes of deformation are ejection, torsion as indicated by the arrows, and base to apex ellipticalization.

deformation, rotation and translation. The simplest mode of deformation is a spherically symmetric radial displacement of the wall, which is related to a decrease in cavity volume and an increase in wall thickness during the ejection phase [1–3]. In the field of displacement of the wall as extrapolated to the cavity, ejected volume is disappearing in a mathematically singular point located at the center of the cavity.

Left ventricular kinematics are described much more accurately when considering additional modes of deformation. One mode of this kind is torsion of the LV around the base to apex axis [4–7], which is quantified by axial–circumferential shear in the wall. Another mode is circumferential contraction in combination with longitudinal extension, often referred to as ellipticalization [8–11]. The combination of the three modes was described in detail in a cylindrical symmetric model of the LV [7, 12, 13]. When describing the kinematics of the left ventricular ejection for the whole LV, the cylindrical model cannot be used, because the axis of the cylinder crosses the LV wall at the apex. The volume near the cylinder axis will be ejected, and this volume cannot be part of the wall (Fig. 1). Obviously, the shape of the LV is neither spherical nor cylindrical, but more that of a prolate ellipsoid with a distinct base to apex axis.

To describe stresses and strains in the fibers of the wall during the ejection phase, three functional modes of deformation can be recognized. The mode of ejecting volume from the cavity is needed for pumping. The modes of torsion and ellipticalization are related to equilibria of forces, and play an important role in transmural evening of fiber stress and strain.

For the torsion and ellipticalization modes, the mechanical equilibrium is described by the state of minimum energy. A deformation mode with amplitude k of the kinematic parameter describes a pattern of displacement $\vec{u}$ as a function of the position $\vec{x}$ in the wall. Then for a small change in stored mechanical energy ΔE in the fibers it holds:

$$\Delta E = \int_{\text{wall}} \sigma_f \Delta e_f dV \quad ; \quad e_f = k\ f(\vec{u}(\vec{x})) \tag{1}$$

where σ_f and e_f indicate fiber stress and fiber strain, respectively. The energy changes as a function of kinematic parameter k. At equilibrium:

$$\frac{\partial E}{\partial k} = \int_{\text{wall}} \sigma_f \frac{\partial e_f}{\partial k} dV = 0 \tag{2}$$

The angle β between fiber direction and circumference at location $\vec{x}$ determines the relation between displacement and fiber strain (right part Eq. (1)). For the equilibria of torsion and ellipticalization it holds:

$$\int_{\text{wall}} \sigma_d \sin\beta \cos\beta \, dV = 0 \qquad \int_{\text{wall}} \sigma_f (1 - 3\sin^2\beta) \, dV = 0 \qquad (3)$$

In the present study, the functional modes of deformation are reconstructed by following the motion of radiopaque markers, inserted in the wall of the LV [14, 15].

To follow the motion of the wall of the LV, six modes of motion have to be added, in addition to the functional modes of deformation, to describe rotation and translation of the whole LV in the three-dimensional (3D) space [16]. Besides, due to gravitational forces, the LV deforms asymmetrically during diastole. The related pattern of deformation has been described by four asymmetric modes, related to deformation of a sphere to an ellipsoid: e_{xx}, e_{yy}, e_{xy}, e_{yz} and e_{zy}, where the z-axis coincides with the long axis of the LV [17]. The three functional motion modes are distinct from the ten other modes, the latter of which are not functionally related to the ejection of blood.

EXPERIMENTS

In five anesthetized open chest dogs, 14–20 radiopaque markers were implanted in the wall of the LV. Stainless steel markers (1.5 mm OD) were inserted at three equally spaced parallel short axis cross-sections in the anterior, lateral and posterior part of the free wall of the LV. The markers were inserted in pairs, one just below and the other 7 mm below the epicardial surface. One or two markers were positioned in the septal wall through the right ventricle. For identification of the anatomical base to apex axis, two ring-shaped markers were attached to the apical dimple and to the invagination between the aortic root and the left atrium. The positions of the markers were recorded by biplane cineangiography with 90 frames per second. In each projection the position of the markers was detected by computerized image analysis. The 3D location of the markers was obtained by pairing the marker images in both projections [14, 15]. Using a least squares method, the kinematic model was fitted to the measured motion of the markers [16].

Given the LV in the reference state, the location of the kinematic center and the direction of the torsion axis are defined by five parameters. To start the motion analysis, the configuration of the LV at approximately mid-ejection is used as the state of reference. The center and the direction of the axis are estimated on the basis of anatomical landmarks. The 13 kinematic parameters were calculated using a least square method for each frame in time. The total residual sum of the squared distances between the model and the measured marker positions were further minimized by varying the location of the center and the orientation of the torsion axis in the reference state. Experimental results on patterns of deformation were compared with the simulated deformation of the LV in a cylindrically symmetric model of left ventricular mechanics [7].

RESULTS

Generally, a good and stable numerical convergence was obtained in the experiments, resulting in a standard deviation of ± 0.3 mm between measured and calculated marker positions. Stable estimates were obtained of the 13 parameters for each frame and the location and orientation of the coordinate system in the reference configuration. In the

control state, the torsion axis was found to be close to the anatomical base to apex axis. The center of the torsion axis crosses the equatorial plane of the LV at a distance of 4 ± 2 mm (mean ± sd), displaced towards the lateral aspect of the free wall.

Figure 2 shows a control beat example of the time course of the seven kinematic parameters which are related to deformation. During the ejection phase, LV cavity volume decreased by a 0.14 fraction of wall volume. Torsion, as expressed by axial–circumferential shear deformation in the wall (k_2), increased by 0.113 radians. Axial shortening was a fraction of 0.04 less than circumferential shortening, resulting in ellipticalization of the LV during the ejection phase. Interestingly, the parameters k_4–k_7 were practically constant during the ejection phase, indicating a minor asymmetric deformation. These latter four parameters vary significantly in diastole, indicating the effect of gravity on the shape of the diastolic ventricle in the open chest.

Figure 3 represents the control beats in five experiments. Left ventricular pressure is plotted as a function of the ratio of cavity volume to wall volume. Furthermore, one loop has been added, describing the pressure–volume plot as generated by a mathematical model of LV mechanics. The bold parts of the curves reflect the relation during the ejection phase. In the control situation in the various experiments, the loading states appeared to be quite different. The end-diastolic ratio of cavity volume to wall volume ranged from 0.22 to 0.49, and the end-systolic ratio ranged from 0.12 to 0.29.

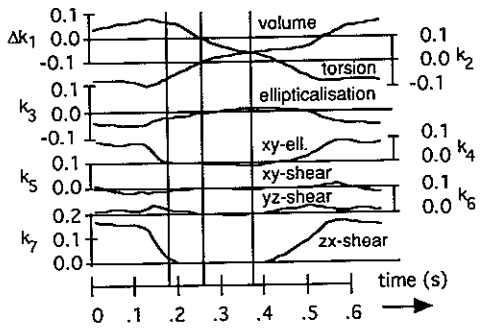

Figure 2. The time course of kinematic parameters related to deformation of the LV during the cardiac cycle. The vertical lines indicate the beginning of ejection, the reference frame of deformation, and the end ejection, respectively. The parameter Δk_1 is associated with changes in ventricular cavity volume, k_2 with torsion, k_3 with the ratio of axial length to diameter, k_4–k_7 with asymmetric linear (shear) deformation. The positive x–,y– and z–directions point to the right, the posterior side, and the apex, respectively.

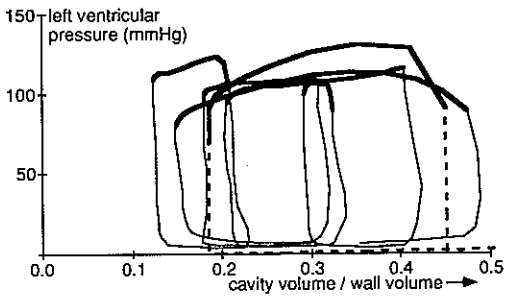

Figure 3. Left ventricular pressure as a function of normalized LV volume. The bold parts of the curves indicate the ejection phase. The dashed line refers to the results of a computer simulation.

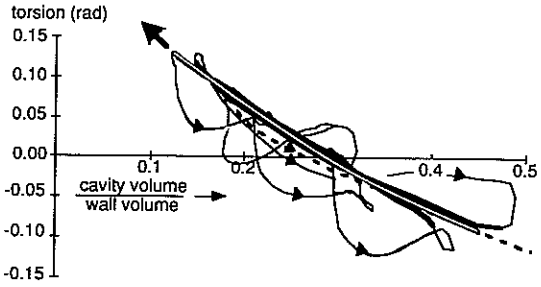

Figure 4. Torsion as a function of normalized LV volume. The bold parts of the curves indicate the ejection phase. The dashed, and partly white line refers to the results of a computer simulation.

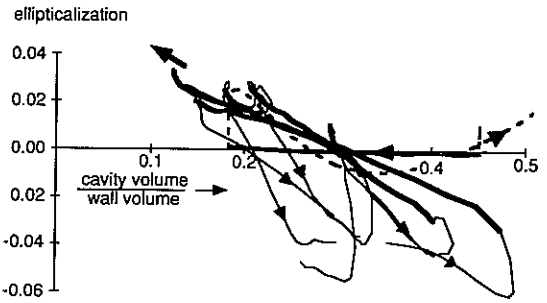

Figure 5. Ellipticalization as a function of normalized LV volume. The bold parts of the curves indicate the ejection phase. The dashed line refers to the results of a computer simulation.

In Figs. 4 and 5, representing the control beats in five experiments, the torsion and ellipticalization are plotted as a function of the ratio of cavity volume to wall volume, respectively. Included in each figure is a curve generated by computer simulation of LV wall mechanics. The bold parts of the curves reflect the relation during the ejection phase. All systolic curves were forced to cross the normalized volume axis at 0.3 by appropriate vertical shift of the individual curve. Strikingly, all systolic torsion–volume curves appear to coincide in one unique curve, which appears to be identical with the curve generated by the simulation. The systolic ellipticalization–volume curves are more different from one experiment to another. During diastolic filling, both the torsion and ellipticalization curves are individually very different.

DISCUSSION

Normal motion of the wall of the LV can be described quite accurately by the 13 motion modes. Three deformation modes are needed for the ejection of blood during systole. Four additional modes reflect asymmetric deformation, which are predominantly important during the diastolic phase. The six rigid body motion modes are not evaluated. When comparing different hearts, the pattern of systolic deformation appears to be quantitatively similar in all experiments after proper normalization. All motion modes are evaluated as a function of the dimensionless ratio of cavity volume to wall volume.

Normally, the axis of the LV is determined on the basis of anatomical landmarks. The anatomical landmarks used in the present study are only an initial estimate of this axis. The final location and direction of the torsion axis is calculated on the basis of the motion of the markers by numerical iteration. The torsion axis appears to be close to the anatomical axis, albeit displaced approximately 4 mm towards the lateral free wall of the LV.

Torsion is expressed by the axial–circumferential shear in the wall. In this definition the circumferential–axial shear is supposed to be zero because of rotational symmetry of the pattern of deformation as related to torsion. In order to obtain the symmetric shear component as related to the deformation of the myocardial material, torsion should be halved. Torsion appears to have a unique quantitative relationship with normalized volume. Moreover, this relation is identical to the relation predicted by an earlier theoretical model of the LV mechanics [7]. The theoretical model was based on the equilibrium of torsion and axial force; torsion was indicated to be a means to even out transmural differences in the strain of the myocardial fibers. Because the model and all control experiments have the same relation between the torsion and the normalized volume, we believe that the basics of the mechanics of the LV are now quite well understood.

The mode of ellipticalization does not relate to the normalized volume as uniquely as the torsion, and the differences between experiments are larger. Furthermore, the ellipticalization–volume curve predicted by the model [7] does not fit the experiments as well as in the case of torsion. Thus, the role of ellipticalization of the LV is not well understood. According to previous model studies, ellipticalization is related to the equilibrium of axial force, which is partly determined by the forces in the mitral valve papillary muscle system [18, 19].

In the present study, general relations are found between dimensionless quantities such as the ratio of cavity volume to wall volume, torsion and ellipticalization. The amplitude of changes in these parameters during the ejection phase depends on the hemodynamic state, which, as shown in Fig. 2, vary considerably with the large variations in the normalized cavity volume in the control state. During ejection, changes in these parameters occur simultaneously. The ratio of changes in these parameters during ejection appears to reflect the pattern of motion, and is much less dependent on hemodynamic load than on the changes in these quantities themselves. Recognition of the motion pattern is important in characterizing the mechanical function of the LV. That means that measuring a change in one parameter such as torsion is not as valuable as the measurement of the ratio of torsion to a change in the normalized volume. It would seem that for clinical applications it would be more useful to stress the search for a ratio of changes of the parameters than to analyze the change of one individual parameter.

In diastole, the relation between the various motion modes is variable. Evidently, the analysis of diastolic motion is much more complicated than systolic motion. No general patterns in diastolic motion could be recognized so far. Because of the large variations in diastolic motion patterns, normal and abnormal diastolic motion cannot be differentiated as easily as in systolic motion. It is to be expected that recognition of aberrant ventricular motion can be based best on data obtained in systole.

CONCLUSIONS

The motion pattern of the LV in the normal open chest canine preparation can be measured by using radiopaque markers inserted in the wall of the LV. Evaluation of systolic motion requires three functional modes: the ratio of cavity volume to wall volume, torsion and ellipticalization. Four additional modes are related to asymmetric deformation

of the ventricle, which mainly occurs during diastole. Finally, six rigid body motion mode are needed. The location of the center of ejection and the orientation of the torsion axis are determined on the basis of the motion of the markers in the wall independent of anatomical landmarks. A unique relation is found during systole between the normalized cavity volume and torsion. This relation is universal to all LVs, and is identical with the prediction of our mathematical model [7]. The patterns of deformation in diastole seem less universal and much less reproducible.

DISCUSSION

Dr. E. Ritman: How was this work done?

Dr. T. Arts: An open chest canine dog in the supine position in a bi–plane x–ray set–up.

Dr. E. Ritman: That bothers me somewhat; within the closed chest with the lung and no pneumothorax, the epicardial surface of the heart is constrained to the extent that it can not move as much as it can in the open chest and that may alter the motion of your heart quite a bit.

Dr. T. Arts: I agree. Especially in the diastolic phase, because then the heart is very deformable. It is lying there in the open air, so it flattens with gravity. I believe that in the closed thorax the shape will not change that much. At the beginning of systole it jumps very quickly into some systolic state. Then the heart feels very stiff, and shape is determined by the internal forces.

Dr. E. Ritman: I think it is sort of chicken and egg problem here. I wonder if all the muscle does is to contract with a certain strain and then the heart twists. It has no option. It is not as though there is somebody in there saying "Let's twist" so that the strains are equal. It may be just the opposite way around.

Dr. T. Arts: According to our models, the distribution of strain is a result related to the basic physics of mechanics. I would not say it is proven, but there is some evidence that it is correct.

Dr. R. Beyar: You suggest that diastolic deformation is variable and I disagree with that. It may be variable in your experiments, but isovolumic relaxation has been clearly shown by a lot of methods to be very consistent. These include the multiple markers method, human data with MRI, dog data with MRI and our recent apical twistometer. All consistently show a pattern of rapid untwisting during isovolumic relaxation.

Dr. T. Arts: As far as I can see, it is the jump from the systolic to the diastolic torsion curve as a function of cavity volume. This jump is indeed always downward, that is right. But if you look more carefully, the distance over which it jumps is not really well defined. I am not so sure about it. In systole you should think of a variance in the order of a few percent and in diastole the recoil may be on the order of 20–30%, and with a much larger variance.

Dr. R. Beyar: You have a spread of the load, which may explain it in these dogs.

Dr. L.E. Ford: Your model is intended to account for the possibility that all of the fibers shorten by the same amount. The available data on this point are a little less than ideal in that they have been obtained in microscopic studies of muscle taken out of a relaxed ventricle. These kinds of marker studies would enable you to see how much shortening actually occurs during ejection in the muscles at various layers of the heart. Have you analyzed the data to see if in fact the strain is equal through the heart wall.

Dr. T. Arts: This is our largest problem. In trying to relate endocardial and epicardial shortening, you should compare it with the diastolic heart. In diastole there is nearly no torsion. At least, the torsion is completely different from what you find during systole. You can not use the studies that are done *in vitro*. There have been several studies which looked into deformation in the systolic phase. Hunter and Douglas have recently found *[Rodriguez E, Hunter W, Royce M, Leppo M, Douglas A, Weisman H. A method to reconstruct myocardial sarcomere lengths and orientations at transmural sites in beating canine hearts. Am J Physiol 1992; 263: H293–H306]* that shortening is indeed quite homogeneously distributed from the endocardial to the epicardial layers, if you consider the interval of the ejection phase. You should not start at the end of diastole but immediately after the jump at the beginning of systole.

Dr. L.E. Ford: Could you analyze your data to see whether in fact the markers were moving towards each other by the same amount in the epicardium and endocardium?

Dr. T. Arts: They probably do, along the fiber orientation probably they do. The problem is that I do not know the fiber orientation.

Dr. E. Yellin: Did your model predict that there would be no base to apex shortening?

Dr. T. Arts: No. The model says that axial shortening and midwall shortening should be similar. That means that no change in shape is expected. Experimentally, a little change was found so the shape factor does not fit our model. I think it is fair to show this.

Dr. Y. Kresh: I am convinced that sarcomere length changes are not sufficient to account for the global chamber ejection fraction. There must be another mechanism to facilitate an ejection fraction of 60–70%. I am not saying your explanation of regional shortening does not account for it; there must be additional components that will dislocate or remove volume from the chamber other than straight sarcomere length changes.

Dr. T. Arts: No. Sarcomere shortening is of the order of 10% over the whole ejection phase. Then the ejection fraction is 50–70%.

Dr. Y. Kresh: It will not work if there is no torsion.

Dr. T. Arts: Even then I would also get it, but the distribution of sarcomere shortening from the endocardial to epicardial layers would not be even. The average of sarcomere shortening would not be affected.

Dr. J.K–J. Li: Can you indicate where the maximum velocity of shortening is?

Dr. T. Arts: The velocity of shortening is maximum close to where the aortic flow is maximum. This point also closely matches the point where the rate of torsion is maximum.

Dr. E. Yellin: The whole model is predicated on shortening. Shortening takes place everywhere and that is what ejects volume. There is lots of experimental evidence, and certainly there is evidence conceptually, that blood has to leave the ventricle on its own momentum; late in systole some of the blood is leaving the ventricle on its own momentum. Therefore there could be a volume change in the ventricle without shortening, due to shape change. In other words, shortening could completely stop and blood could still leave the heart because it has inertia.

Dr. T. Arts: Our data do not show that. If you look at the change in volume, it stops where flow crosses zero and then it looks more or less stable until the end of ejection. Then the aortic valve closes.

Dr. E. Yellin: If blood leaves, the volume has to decrease. My point is that some of the decrease in volume late in systole is not due to sarcomere shortening, but to a shape change due to blood leaving on its own inertia; it is being sucked out.

Dr. T. Arts: It might be, but we did not measure a shape change. It is part of the pump function and that means that the sarcomeres are shortening. The inertia effect seems not to be related to a change of shape.

Dr. E. Yellin: During the ejection phase the sarcomeres stop shortening, blood leaves the heart, and whatever you see after that point is a shape change, because the sarcomeres have stopped shortening.

Dr. T. Arts: We did not measure a shape change at the end of the ejection phase. In other experiments we have measured epicardial shortening with a video technique. Then you see that the length of the fibers follows very well the pattern of volume change. The sarcomeres stop shortening at the moment of closure of the aortic valve.

Dr. E. Yellin: Let me put it in another way. Suppose the sarcomeres stop shortening, and we have a little Maxwell demon who sits at the aortic valve and prevents the valve from closing, and sucks blood out of the ventricle. What would happen?

Dr. T. Arts: You create a vacuum and the fibers will shorten anyway.

Dr. E. Yellin: Yes, you will create a vacuum! And that is why ventricular pressure is less than aortic pressure sometime in mid-systole.

Dr. T. Arts: Yes, but fibers shorten anyway. You do not know if this is active force generation by the fibers, or whether they are shortened by their environment.

Dr. E. Yellin: Interesting point; they could stop shortening, blood could still leave and the ventricle could distort. We can not prove it, but it is worth thinking about.

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