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STIFFENING OF THE CARDIAC WALL BY CORONARY BLOOD VOLUME INCREASE: A FINITE ELEMENT SIMULATION

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ABSTRACT

A porous medium finite element model of the beating left ventricle is used to simulate the influence of the intracoronary blood volume on left ventricular mechanics. The spongy material is composed of incompressible solid (myocardial tissue) and incompressible fluid (coronary blood). The model is axisymmetric and allows for finite deformation, including torsion around the axis of symmetry. The total stress in the tissue is the sum of the intramyocardial pressure, effective passive stress due to myocardial deformation and the contractile fiber stress. The model is able to simulate a full cardiac cycle. Three-dimensional end-systolic deformation computed relative to the end-diastolic state is shown to be consistent with experimental data from the literature. The direction of maximal shortening varied less than 30° from endocardium to epicardium while fiber direction varied by more than 100°. It is shown that the ventricular model exhibits diastolic stiffening following an increase of intracoronary blood volume. End-diastolic left ventricular pressure increases from 1.5 kPa to 2.0 kPa when raising intracoronary blood volume from 9 to 14 ml per 100 g myocardial tissue. The model simulation suggests that the mechanism underlying the increase in end-diastolic pressure at higher coronary blood volumes, is an increase in passive stiffness of the myocardial fibers. This increased stiffness is the combined result of an overall increase in strain in myocardial tissue and the non-linear stress-strain relationship of myocardial tissue.

Keywords
Left ventricle / porous medium / mixture / erectile properties / diastole / coronary perfusion.

INTRODUCTION

The cardiac wall is a complex biological structure composed of different components: muscle cells, coronary vessels, collagen fibers,

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intracellular and interstitial fluid, lymph and blood. For the sake of simplicity, many authors of cardiac models assume the cardiac wall to be a homogenous continuum of solid matter. The aim of this study is to investigate to which extent a more detailed model description of the myocardial tissue, including a solid and a fluid component, is able to describe the stiffening of the cardiac wall by coronary blood volume increase.

METHODS

Material model. We assumed that myocardial tissue was a spongy structure filled with intracoronary blood. The material of which the spongy structure was made and the intracoronary blood were both assumed to be incompressible. Therefore, changes in volume of the solid fluid mixture were equal to the amount of blood being squeezed out or sucked in. The stress in the mixture was the sum of the intramyocardial pressure p (present in both fluid and solid), effective passive stress due to the deformation of the porous structure and contractile fiber stress. As the collagen weave is three-dimensional, we assumed that the passive effective stress was three-dimensional. The contractile fiber stress, however, acted only in the fiber direction, which changed across the wall. The contractile stress was time, strain and strain rate dependent. The redistribution of intramyocardial blood in the coronary bed was modelled by Darcy's law: the flow of intracoronary blood was proportional to the intramyocardial pressure gradient. The proportionality constant is the permeability of the medium. In order to account for the changes in vascular resistance when intracoronary blood volume changes, the permeability of the medium was adapted proportionally to the square of the intramyocardial coronary blood volume in the course of the computation.

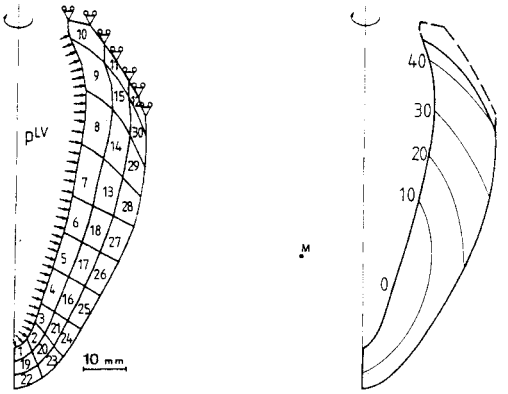

Fig. 1 Left: cross-section of the rotationally symmetric finite element mesh of a canine left ventricle. All elements contain contractile fibers except elements 10, 11 and 12 which represent the annulus fibrosus. Right: in a cross-section of the wall of the left ventricle, the sequence of onset of contraction is simulated to radiate from a point M (time in ms).

Numerical approach. The myocardial wall was subdivided into thirty ring-shaped elements (fig. 1). Each element had eight nodes. The change in position of each node relative to the reference situation was described by a radial, axial and circumferential displacement at any time during the cardiac cycle. Quadratic interpolation yielded displacements at intermediate points. The intramyocardial pressure field was obtained by linear interpolation from pressure values at the corner nodes. The total number of degrees of freedom was 395 (351 displacements and 44 pressures). finite deformation was accounted for by means of a total Lagrangian

approach. An implicit-explicit time integration scheme was used. Within each time step a modified Newton-Raphson iterative procedure was used to account for the non-linearities included in the model.

The axial and circumferential displacement of the 7 top nodes of the mesh were suppressed. No blood was allowed to cross the endocardial surface. At the endocardial side of elements 1 to 9 a uniform intraventricular p^{LV} was applied as an external load. The loads exerted by the papillary muscles and by the pericardium were neglected. Along the epicardial surface we allowed free exchange of blood between the intramyocardial coronary vessels and the epicardial coronary vessels. Initiation of contraction was not simultaneous for all sarcomeres (fig. 1). The depolarisation wave moved from endocardium to epicardium and from the apical region toward the basal region. The wave needed about 40 ms to reach the whole left ventricular wall. The initial permeability of the porous medium was derived from data on time constants of the coronary circulation and equals 2 $mm^2\ kPa^{-1}s^{-1}$. The initial porosity of the medium (= the percentage of intramyocardial space occupied by coronary blood at 0 kPa perfusion pressure) is 6%.

The transmural variation of fiber angle was derived from experimental data of Streeter and Hanna (1973). The sarcomere model used in the simulations is described elsewhere (Huyghe, 1986). The passive constitutive behaviour of the myocardial tissue was a quasi-linear viscoelastic law with an exponential elastic response, which was fitted to experimental data of van Heuningen et al (1982), and Yin et al (1987).

Three computations were performed. Each computation started with an increase of intracoronary blood volume. The increase equalled 3% of the myocardial volume in the first computation, 5,5% in the second computation and 8% in the third computation. After the increase of intracoronary blood volume, the intraventricular pressure was increased up to 2.5 kPa in all three computations. Finally the third computation was repeated, loading the ventricle only up to 1 kPa, and then consecutive beats were initiated. The duration of the cardiac cycles was 0.55 s.

A commercial post-processing package I-DEAS (Structural Dynamics Research Corporation) produced color-coded plots of different local output variables. To facilitate the comparison of computed strains with experimental strain data, local three-dimensional Green strain tensors were computed with reference to the end-diastolic state and were interpreted in terms of their eigenvalues (the principal strains) and eigenvectors (the principal axes of strain). The three principal strains were ranked from smallest (most negative) to largest (most positive).

RESULTS

Diastolic stiffness. The increase in intracoronary blood volume induced an increase of wall thickness. The pressure-volume curves resulting from the three computations (fig. 2) show that diastolic stiffness increased with increasing coronary vascular volume. At a given left ventricular volume left ventricular pressure increased from 1.5 kPa to 2.0 kPa when raising intracoronary blood volume from 9 to 14 ml per 100 g left ventricle.

The cardiac cycle. Ejection fractions for the three cardiac cycles were 59%, 55% and 54%. The time course of the radial and axial displacement component of all nodes of the mesh is shown in fig. 3.

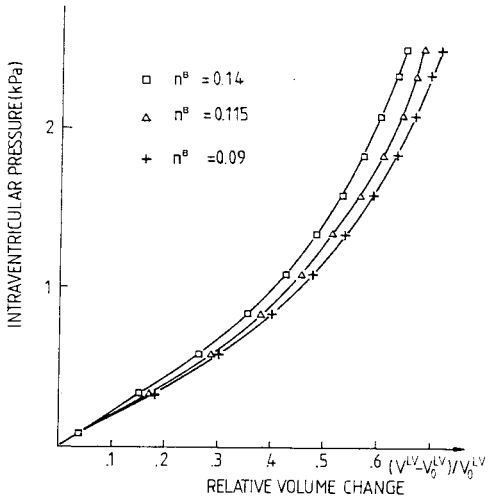

Fig. 2 Simulated pressure-volume relations of the passive left ventricle at different levels of intramyocardial blood volume. The ventricular wall stiffens at increased intramyocardial blood volume. V_0^{LV} and V^{LV} are the initial and current intracavitary volume of the left ventricular model respectively. n^B is the ratio of coronary vascular volume over total myocardial volume.

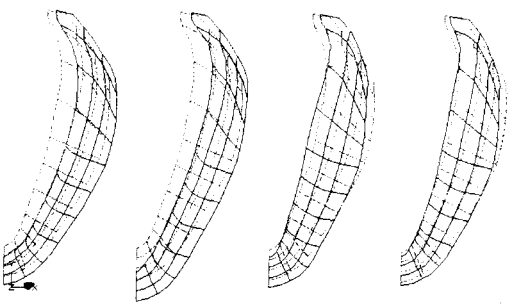

Fig. 3 Simulated successive states of deformation of the left ventricle. A computer generated picture is shown of the deformation of a meridional section of the model (dotted line = reference state, continuous line = deformed state). From left to right: end diastole, beginning and end of ejection and beginning of diastole.

The circumferential displacement component (not shown) showed rotation of the apex relative to the base in counterclockwise direction during the ascending limbs of the ventricular pressure, while the opposite happened during the descending limb. The model computed increasing end-systolic values of the three principal strains with increasing depth (fig. 4).

End-systolic principal strains equalled 0.45; -0.01 and -0.24 at 2/3 of the wall thickness from the epicardium and 0.26, 0.00 and -0.19 at 1/3 of the wall thickness from the epicardium. To analyse the transmural variation in the orientation of the principal strain axes at the end of ejection, we computed the angle between the first principal strain axis (i.e. the axis of maximal shortening) and the circumferential coordinate

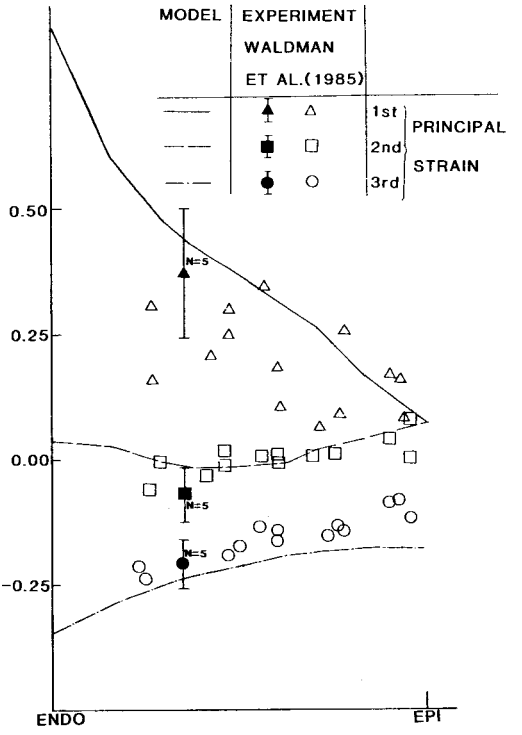

Fig. 4 Transmural distribution of end-systolic principal strains as predicted by the model and measured by Waldman et al (1985). In both model and experiment strains are given with respect to the end-diastolic state.

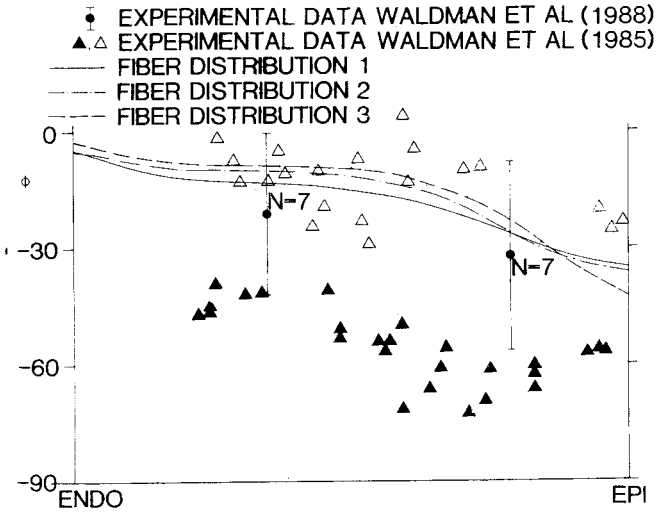

Fig. 5 The angle ϕ is the projection onto the epicardial plane of the angle between axis of maximal shortening and the circumferential direction. The variation of angle ϕ with depth is plotted for three fiber angle distributions, defined in table 1. The two sets of experimental data (▲ and △) are from two animals representing the range observed in five dogs by Waldman et al. (1985).

direction. The projection of this angle on the epicardial tangent plane is refered to as the angle ϕ and is plotted as a function of depth at the interface of the elements 6, 18, 27 and the elements 7, 13, 28 (fig. 5). The angle ϕ shifted from -37° epicardially to -6° endocardially.

DISCUSSION

This study shows that (1) a biphasic model of left ventricular mechanics is able to compute an increase of diastolic stiffness by coronary blood volume increase, provided that the non-linear nature of the constitutive behaviour of myocardial tissue is taken into account and (2) the axisymmetric finite element model computes systolic triaxial strains consistent with experimental data from the literature.

Diastolic stiffness. When using quasi-linear viscoelasticity, stiffness increases with increasing coronary vascular volume (fig. 3). This result is consistent with experimental data of Olsen et al. (1981) and Vogel et al. (1982) (fig. 6).

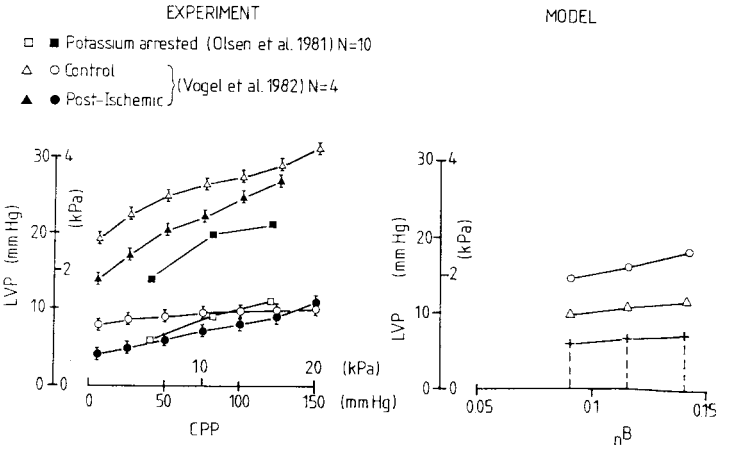

Fig. 6 The left panel shows the dependency of end-diastolic left ventricular pressure (LVP) on the coronary perfusion pressure (CPP) at a given cavity volume. The right panel shows model results of left ventricular pressure as a function of intracoronary blood volume at constant cavity volume. Stiffening of the cardiac wall by increase of coronary vascular volume is more pronounced at higher cavity volumes, in both experiment (left) and model (right).

On the basis of the experimental quantification of the relationship of coronary perfusion pressure and intracoronary blood volume by Morgenstern et al. (1973), we infer that the three values of intracoronary blood volume chosen in fig. 3 correspond with coronary perfusion pressures of 6, 11, and 14 kPa. This implies that the model predicts that at constant cavity volume, left ventricular pressure increases from 1.5 kPa to 2.0 kPa when raising perfusion pressure from 6 to 14 kPa. The same shift of coronary

perfusion pressure causes an increase in left ventricular pressure from 1.5 to 2.4 kPa in potassium arrested hearts according to the experimental data of Olsen et al. (1981), while Vogel et al. (1982) find an increase of left ventricular pressure from 1 to 1.3 kPa and from 3 kPa to 3.8 kPa in intact hearts indicating that model results are within the range of values measured experimentally. The increase in wall thickness induced by the increased intracoronary blood volume is consistent with the experimental data of Morgenstern et al. (1973). One might be tempted to attribute the shift of the pressure-volume curve to the left in fig. 3 to the increase in wall thickness, on the basis of Laplace's law. However, it is not evident that Laplace's law applies to biphasic materials. In a single phase solid material, increase in thickness of the shell induces an increased stiffness of the shell, because more material is available to take up shell forces. In the case of a solid-fluid mixture, adding more fluid to the mixture, does not result in additional material to take up shell forces. Tensile forces can only be borne by the solid. The increase in intracoronary blood volume resulted in increased wall thickness, but not in increased stiffness when we replaced the quasi-linear viscoelastic law by isotropic linear elasticity while maintaining Darcy's law as a description for the redistribution of intracoronary blood. These results show that stiffening of the diastolic left ventricle by coronary blood volume increase should not be interpreted in terms of Laplace's law, but rather as the combined result of an overall increase in strain in myocardial tissue and the non-linear stress-strain relationship of the myocardial tissue.

The cardiac cycle. The model computes increasing values of strain with increasing depth. Many experimental data from the literature point in the same direction. In fig. 4 we have used the experimental data of Waldman et al. (1985) to assess the transmural distribution of principal strain as predicted by the model. Although the direction of maximal stress almost coindices with the fiber direction, the direction of maximal shortening (i.e., the third principal strain axis) does not. Across the wall the computed direction of maximal shortening does not vary nearly as much as the muscle fiber direction (fig. 5). This finding is consistent with experimental data of Prinzen et al. (1984) and Waldman et al. (1985). The latter investigators found that the above defined angle ϕ of maximal shortening equaled $-22\pm21°$ in the inner half of the wall ($65\pm9\%$ of the wall thickness from the epicardium). A common feature of the model prediction and Waldman's data is the progressive rotation of the principal axis of shortening towards the circumferential direction with increasing depth.

CONCLUSION

An axisymmetric two-phase finite element model is used to simulate myocardial deformation during the cardiac cycle. Computed transmural strain distribution is in agreement with experimental data from the literature. The model indicates that the increase in diastolic stiffness by increase of coronary vascular volume should be interpreted as a combined effect of an overall increased strain in the myocardial fibers and the non-linear stress-strain relationships of the myocardial tissue.

Question from the audience: Isn't axisymmetry a rather rough approximation of the real geometry of the left ventricle?

Huyghe: We chose for an axisymmetric model in order to reduce computation time. This approximation is indeed a limitation of this model. In december, our group will present a three-dimensional model of the left ventricle at the Winter Annual Meeting of the ASME.

Question from the audience: How do you analyse torsional deformation with an axisymmetric model?

Huyghe: The finite element code that we use has been written specially for finite deformation including torsion. Except a radial and axial displacement, there is also a circumferential displacement, which is also axisymmetric.

Oddou: There is an essential difference between the porous medium approach of your group and our group, because fluid in your model is the coronary blood, while in our analysis it is the interstitial fluid.

Huyghe: This is very right. The two phenomena have also very different time constants and this is why we feel it is acceptable to neglect interstitial fluid flow in our analysis.

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