Transmural course of stress and sarcomere length in the left ventricle under normal hemodynamic circumstances

Citation for published version (APA):

Reneman, R. S., Arts, M. G. J., & Veenstra, P. C. (1980). Transmural course of stress and sarcomere length in the left ventricle under normal hemodynamic circumstances. In *Cardiac dynamics* / Ed. J. Baan, A.C. Arntzenius, E.L. Yellin (pp. 115-122). (Developments in cardiovascular medicine; Vol. 2). Nijhoff.

Document status and date:

Published: 01/01/1980

Document Version:

Publisher's PDF, also known as Version of Record (includes final page, issue and volume numbers)

Please check the document version of this publication:

- A submitted manuscript is the version of the article upon submission and before peer-review. There can be important differences between the submitted version and the official published version of record. People interested in the research are advised to contact the author for the final version of the publication, or visit the DOI to the publisher's website.
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- The final published version features the final layout of the paper including the volume, issue and page numbers.

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2.4. TRANSMURAL COURSE OF STRESS AND SARCOMERE LENGTH IN THE LEFT VENTRICLE UNDER NORMAL HEMODYNAMIC CIRCUMSTANCES

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1. INTRODUCTION

In this paper the dynamic behaviour of mechanical stresses and strains in the wall of the left ventricle is described. Though several investigators are rather optimistic (1), direct determination of stresses in the wall of the left ventricle is difficult and unreliable (2). A better approach is calculation of these stresses by the use of a mathematical model of the mechanics of the left ventricle (3). Following this principle, several investigators (2, 4, 5) have computed wall stress from left ventricular pressure, assuming a certain geometry of the left ventricle and certain mechanical properties of the wall. However, none of these models can be used to study the dynamic behaviour of the stresses in the wall of the left ventricle since these models are based on unrealistic approximations, such as isotropic myocardial material or inadequate geometry, while usually fibre orientation in the wall of the left ventricle and physiological contractile behaviour of the myocardial material are not taken into account. Therefore, a new model was developed in which all of these factors are considered (3).

2. DESIGN OF THE MATHEMATICAL MODEL OF THE LEFT VENTRICLE

In designing a mathematical model of the mechanics of the left ventricle, simplifications concerning the characteristics of the heart are required. Each simplification is a compromise between accuracy and simplicity. Following this principle the following simplifications were made.

- Only the left ventricle was simulated.
- The left ventricle is a thick-walled cylinder composed of eight concentric cylindrical shells (Figure 1). The relation between left ventricular pressure and wall stress is most realistically described by an ellipsoidal geometry. Cylindrical geometry, however, approximates the realistic relation closely, i.e. within 10%, and better than if spherical geometry is assumed (3).
- The muscle material is taken to be anisotropic, because experiments have shown that assuming isotropic properties of the myocardial wall, unrealistic findings are obtained (3,6). Anisotropy is introduced by assuming

Figure 1. Schematic representation of the geometry of the left ventricle. The ventricle is simulated by a cylinder. Subdivision of the thick-walled cylinder in a number of cylindrical shells and changing fibre orientation are indicated.

that the myocardial tissue consists of a contractile fibre structure embedded in a soft, incompressible material (6).

- The contractile properties of the fibre structure were derived from experimental data on papillary muscle (7,8). These properties were simplified in a mathematical model which calculates the velocity of sarcomere shortening from the instantaneous state of activation and the tensile stress borne by the fibre structure (3).
- The fibre orientation changes across the ventricular wall according to the findings of Streeter and Hanna (9).
- Spread of onset of muscle contraction across the wall is assumed to occur only in a radial direction, and is based on the electrical activation pattern found by Durrer et al. (10).
- The aortic valve and aortic input impedance were simplified to a network (11) which is represented diagrammatically in Figure 2.

The mathematical basis of the model is a system of differential equations.

Figure 2. Network which represents the aortic input impedance including the aortic valve.

Thus the time derivatives of a number of characteristic quantities were calculated, so that the increment of these quantities during the following increment in time could be computed. In this way the values of the characteristic quantities were computed at intervals of 2 msec. All quantities of physiological interest incorporated in the model were derived from the set of characteristic quantities.

The instantaneous state of deformation of the myocardial material was calculated from the volume of the inner cavity and the cylindrical shells and from the height and torsion angle of the cylinder. The height of the cylinder (Figure 3) represents the base-to-apex distance. The torsion angle is defined as the angle over which the upper cross-sectional surface of the cylinder is rotated around the axis with respect to its lower cross-sectional surface (Figure 3). This angle represents the angle of rotation of the base with respect to the apex around the axis of the left ventricle (3). The height and torsion angle are calculated from equilibria of forces and torques in the upper cross-sectional surface of the cylinder. The axial force exerted by the left ventricular pressure is in equilibrium with the total force due to axial stresses in the wall of the cylinder. The torques acting in the upper surface of the cylinder due to tangential shear stresses are in equilibrium too. Both equilibria result in a set of two equations, that can be solved for height and torsion angle.

3. SIMULATION OF A CARDIAC CYCLE

In order to simulate a cardiac cycle, values of several parameters had to be substituted in the mathematical relations describing the model. Values of many parameters were obtained from experimental results described in the

Figure 3. Definition of the height h and torsion angle α of the cylinder. The arrow denotes the rotation of the upper surface over an angle α due to the deformation of the cylinder.

literature such as end-diastolic ratios of left ventricular wall thickness and height to internal radius (12), transmural distribution of fibre orientation (9), sarcomere length (9) and sequence of depolarization (10) and, finally, contractile properties of cardiac muscle (7,8). To be able to compare the simulation with an animal experiment, the values of several parameters, such as heart weight, aortic input impedance, end-diastolic left ventricular pressure, and heart rate, were obtained from the findings in that particular experiment.

A simulation of a normal cardiac cycle is shown graphically in Figure 4. The time course of left ventricular and aortic pressure and aortic volume flow agree fairly well with results obtained from animal experiments under comparable circumstances (Figure 5). In the simulation the cylinder lengthens 2.3% and the inner radius shortens 1.1% during the isovolumic contraction phase. During ejection, height and radius decrease 6.3% and 23%, respectively (Figure 4).

At the beginning of the isovolumic contraction phase, the torsion angle decreases. Just before opening of the aortic valve, the torsion angle reaches a minimum and subsequently increases approximately linearly during ejection. The angle reaches a maximum during the relaxation phase whereupon it decreases rather rapidly until the diastolic level is reached. It is remarkable that no discontinuities in the time course of the torsion angle are present when the aortic valve opens or closes.

Figure 4. Results of a simulation of a normal cardiac cycle: p_{ao} = aortic pressure; p_{lv} = left ventricular pressure; q_{ao} = aortic volume flow; h = height of the cylinder; r_{iv} = inner radius of the cylinder; α = torsion angle of the cylinder; σ_1 = tensile muscle stress ($t = 120$ msec); w = generated mechanical energy per unit of volume per beat.

Figure 5. Experimental results of a normal cardiac cycle (dog 25 kg).

Quantitative data concerning the time course of base to apex distance and radius are available from animal experiments of Rankin et al. (Figure 6). In their experiments the increase of the base-to-apex distance during the isovolumic contraction phase was 2.1%. During ejection this distance and the inner radius decreased 6.2% and 22%, respectively. These findings are in excellent agreement with the results obtained from the model, in which the height and inner radius of the cylinder, respectively, represent the base-to-apex distance and the inner radius of the left ventricle. The behaviour of the torsion angle in the model cannot be evaluated since to our knowledge no information is available about the torsion angle in animal experiments.

From the model several physical mechanisms can be understood. Since

Figure 6. Time course of dimensions of the heart, aortic volume flow and left ventricular pressure in a normal cardiac cycle, as measured by Rankin et al. (13).

the results of the model agree fairly well with several findings in animal experiments, some of the underlying mechanisms of the model may be extrapolated to the *in vivo* situation. Examples of such mechanisms are described below.

Changes in dimensions of the cylinder during isovolumic contraction can be explained as follows. The innermost subendocardial layers are activated first. The fibres in these layers are directed in such a way that the influences of axial and tangential stresses on the geometry are approximately in equilibrium. However, proceeding activation of the myocardium involves contraction of more tangentially directed fibres, resulting in a decrease of inner radius and concomitant lengthening of the cylinder during the isovolumic contraction phase.

Because of the spiral course of the subendocardial muscle fibres around the ventricular cavity, early shortening of the subendocardial sarcomeres in the model causes twisting of the ventricle around its axis early in the isovolumic contraction phase. This is shown by a decrease of the torsion angle. Due to a reverse spiral course of the subepicardial muscle fibres, those fibres are stretched by this twisting. Later in the isovolumic contraction phase contraction of the subepicardial layers has developed to such an extent that the stresses generated by these layers are large enough to prevent further twisting. Then the torsion angle reaches a minimum. Without torsion, subendocardial sarcomere shortening during ejection would be much larger than subepicardial sarcomere shortening because of the thick-walled behaviour of the cylinder. An increase of the torsion angle without ejection lengthens subendocardial sarcomeres and shortens subepicardial sarcomeres. In the model, the torsion angle appears to increase during ejection in such a way that both effects cancel and transmural differences in velocity of sarcomere shortening are small.

In the simulation transmural differences in tensile stress as well as sarcomere length are small at the beginning of the ejection phase. The differences in tensile stress (Figure 4) and velocity of sarcomere shortening remain small throughout the ejection phase. Therefore, at the end of ejection transmural differences in sarcomere length and generated mechanical energy of shortening per unit of muscle volume are small too.

The small differences in tensile muscle stress across the wall during systole can be explained by the character of the transmural course of the fibre orientation. The transmural course of tensile muscle stress is mainly a result of equilibria of forces and torques in the upper surface of the cylinder. Layers with a fibre angle close to 45° and close to -45° counterbalance through the equilibrium of torques in the upper surface of the cylinder. Layers with mainly axially and tangentially directed fibres counterbalance through the equilibrium of axial forces in the upper surface of the cylinder. Extrapolated to the real ventricle this means that the layers between the center and subepicardium counterbalance with the subendocardial layers

through the equilibrium of torques, whereas the center layers counterbalance the subepicardial layers through the equilibrium of axial forces. The transmural course of the fibre orientation determines the thickness of the layers, which are involved in these equilibria, and thus determines the transmural distribution of tensile muscle stress. Since the transmural course of the fibre angle changes to a minor degree during the cardiac cycle, tensile stress remains uniformly distributed during the whole systolic period.

SUMMARY

In a mathematical model of the dynamics of the left ventricle, the ventricle is simulated by a thick-walled cylinder composed of eight concentric subcylinders. The orientation and the sequential activation of the muscle fibres across the ventricular wall are considered per subcylinder. The relation between tensile muscle stress, sarcomere length, velocity of sarcomere shortening, and time are derived from experimental data.

In the model, contraction starts in the subendocardial layers. Because of the spiral course of the subendocardial fibres around the left ventricular cavity, torsion of the left ventricle around its axis and stretching of the subepicardial muscle fibres occur during the isovolumic phase of contraction.

Transmural differences in quantities which are closely related to the state of mechanical loading of myocardial material, such as tensile stress, sarcomere length, velocity of sarcomere shortening and generated mechanical energy per unit of volume are small. Hence, the heart seems to be optimally designed, as far as the process of contraction is concerned.

The validity of the model is demonstrated by the good agreement between the model and animal experiments as far as left ventricular pressure, aortic pressure and volume flow and left ventricular dimensions are concerned. A general conclusion is, that the present model is a useful tool in understanding the dynamic behaviour of the mechanics of the left ventricle.

ACKNOWLEDGEMENTS

This work was supported by a grant from the Foundation for Medical Research FUNGO.

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