The Influence of Cardiac Trabeculae on Ventricular Mechanics

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Abstract

Cardiac trabeculae are cylindrical structures arranged in a complex shape which cover the endocardial surfaces of ventricles. The trabeculae consist of axially arranged cardiac fibers and represent a significant percentage of the ventricular mass (from 12% to 17%). The aim of the project is to study the influence of the trabecular mass on heart performances.

A finite-element model of the left ventricle was developed. To compare different models (with or without trabecular structures) the ventricle was simplified as a truncated ellipsoid and the trabeculae, if present, as cylindrical strands oriented along the ventricular axis direction laying onto the endocardium. The total muscular mass and the intraventricular volume were kept constant in all the models. To model the mechanical behavior of the cardiac tissue, the Holzapfel constitutive relation was chosen. Cardiac fibers were oriented helically in the compact ventricular wall and axially in the trabeculae. Physiologic atrial pressure was set during the ventricular filling phase, while an adequate RCR model was connected downstream the ventricle to simulate the systemic circulation.

The results show a significant role of trabeculae on ventricular behavior: the presence of trabecular mass contributes to the ventricular filling, allowing a higher ventricular stroke volume at the same atrial pressure.

1. Introduction

The human heart evolves in the embryonic stage from a simple tube into a complex structure, composed by four chambers optimized for blood pumping. During heart development, one important event is the emergence of trabeculation in the luminal layers of the myocardium, which enables the cardiac tissue growth in the absence of coronary circulation [1]. In a subsequent stage, there is a compaction of this spongy structure, but an inner layer of trabecular tissue still remains.

In the adult heart, cardiac trabeculae appear as cylindrical structures which cover the endocardial surfaces of both ventricles. They are characterized by an axial orientation of cardiac fibers and are arranged in a complex shape, preferably arising from the free wall and insert into the atrio-ventricular ring.

The trabecular mass represents a significant percentage of the ventricular total mass; in healthy subjects, this percentage has been estimated to vary from 12 to 17% of the total ventricular muscular mass [2,3].

Left ventricular trabeculae are frequently seen during echocardiographic examinations, and even if there is not a universally accepted criteria, a value of trabecular mass higher than 20% of ventricular mass is considered an index of left ventricular non compaction, a primary cardiomyopathy of genetic origin that can lead to heart failure, trombo-embolism and arrhythmia.

Despite these findings, the literature lacks of studies about the role of trabecular mass on heart behavior.

The aim of this work is to understand the influence of ventricular trabeculae on cardiac performances.

2. Materials and methods

To understand the influence of cardiac trabeculae on heart mechanics, a finite element model of the left ventricle was developed within the finite element framework provided by ABAQUS (ABAQUS®, SIMULIA Corp.).

In particular, the purpose is to compare the ventricular behavior in the presence or the absence of the trabecular non-compact layer. Thus, different geometrical models, with or without trabeculae, were designed keeping both the total ventricular muscular mass and the intraventricular volume constant.

2.1. Geometrical model

Two different types of geometry were implemented: a "smooth" and a trabeculated model. In both cases, the left ventricle was simplified as a truncated ellipsoid.

The undeformed configuration of the smooth ellipsoid is characterized by a major axis of 57 mm, a minor axis of 14 mm and a constant wall thickness of 9 mm; thus, the ventricular volume at zero pressure is 43 ml, according to

experimental findings [4].

To design the trabeculated geometry, the 15% of the total muscle mass was changed from compact layer to trabeculae at the endocardium, keeping constant the intraventricular volume. The trabeculae were described as cylindrical strands oriented along the ventricular axis direction, laying onto the endocardium (Fig.1).

Thus, the epicardial surface is the same in both models, while the endocardial layer is characterized by a different muscle mass distribution. Therefore, the wall thickness is not uniform in the trabeculated model: at the trabeculae myocardial wall thickness reaches 10.3 mm; in the inter-trabecular spaces the wall is 7.2 mm thick.

The smooth model was discretized into about 18000 8-node hexahedral elements, while about 61000 8-node hexahedral elements were required for the trabeculated model, duo to its geometrical complexity (Fig.1).

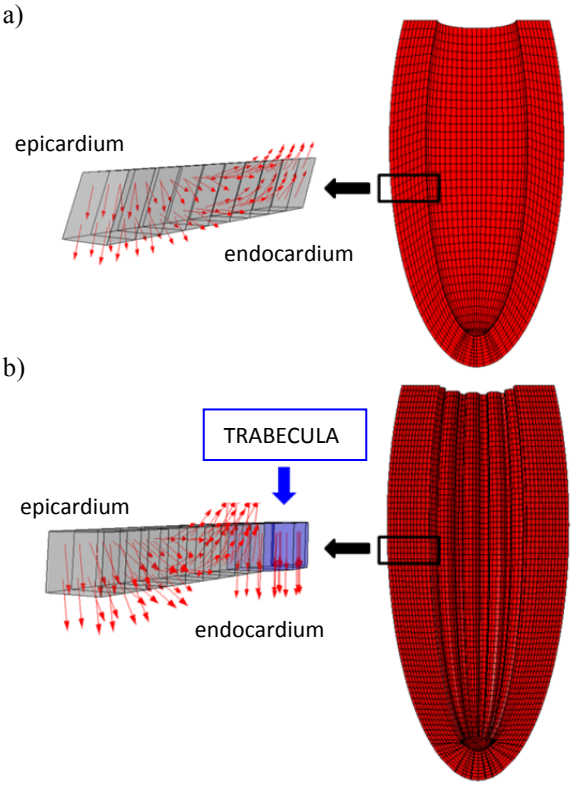

Figure 1. Geometrical model and fiber distribution from epicardium to endocardium in (a) the smooth model and (b) the trabeculated model. The trabecula, with an axial fiber orientation, is highlighted in blue.

2.2. Cardiac tissue modeling

One of the central aspect in the development of a computational model is the identification of the material behaviour. For a ventricular model, two characteristics are important for the model response: a realistic cardiac fiber architecture and an adequate constitutive relation for the myocardium.

Fiber architecture: The left ventricular wall is composed of discrete layers of parallel myocytes, which represent the major part of myocardial volume (~70%), and a specialized connective tissue hierarchy, which connects adjacent muscle fibres.

The orientation of fibres changes with position across the ventricular wall: in the compact layer, the preferential fibres direction is about -80° near the epicardium, rotates to 0° at the midwall and reaches +80° at the endocardium with respect to circumferential direction [5]; in the non-compact layer the fibres follow the trabecular axial direction.

To replicate this fiber arrangement in the ellipsoidal models, a different orientation of fibers was assigned at every "sheet" which composed the compact part of the ventricle using a custom MATLAB routine (MATLAB®, The MathWorks, Inc). Moreover, in the trabeculated model, the fibres were oriented axially in the trabeculae (Fig.1).

Constitutive relation: To simulate the entire cardiac cycle, both the passive and active behaviour of the cardiac fibres have to be modelled.

About the passive material properties**,** the characteristic fibrous micro-structure of myocardium suggests for the single muscle layer of parallel myocytes at least a transversely isotropic behaviour. Indeed, several literature experimental tests have highlighted a higher material stiffness in the myocytes direction with respect to other directions [6]. Thus, a hyperelastic anisotropic consituitve model was chosen (Eq.1-3); in particular, the strain energy potential ($\boldsymbol{\Psi}$) consists of the sum of two terms: the first term relates to an isotropic matrix, which models the extra-cellular connective tissue, while the second term relates to the muscle fibres embedded in that matrix [7]. The direction of the fibers in the reference configuration is characterized by a unit vector $\boldsymbol{A_\alpha}$.

$$\boldsymbol{\Psi} = C_{10}(\bar{I}_1 - 3) + \frac{k_1}{2k_2}(\exp(k_2(\bar{I}_4 - 1)^2) - 1) \quad \text{(Eq.1)}$$

$$\bar{I}_1 = tr(\bar{\boldsymbol{C}}); \qquad \bar{I}_4 = \boldsymbol{A_\alpha} \cdot \bar{\boldsymbol{C}} \cdot \boldsymbol{A_\alpha} \quad \text{(Eq.2,3)}$$

This form of the constitutive law is valid for a single family of perfectly aligned fibers. The other material parameters (C_{10}, k_1, k_2) were chosen such that the diastolic pressure-volume (P-V) relationship of the smooth model fitted a physiological one [8]. Indeed, to avoid the underestimation of the physiological ventricular compliance for the smooth model, it was necessary to scale the mechanical parameters resulting from the fitting of literature experimental tests results [6].

Furthermore, the active behavior of myocytes was implemented. The muscle contraction was obtained by changing the material parameters during the systolic phase; the parameters variation (Fig.2) follows the curve

of the force generated by the muscle fibers secondary to an intracellular calcium variation [9].

The material parameters values during diastole and the maximum value reached during the systolic phase are reported in Table 1.

The maximum parameters values were chosen to obtain a physiologic ventricular torsion peak in the smooth model. Indeed the ventricular torsion, which is the rotational movement of the ventricular apex with respect to the base, reflects the accurate synergic interaction between myocardial constitutive components; a physiologic value of ventricular torsion is fundamental for a realistic ventricular kinematics [10].

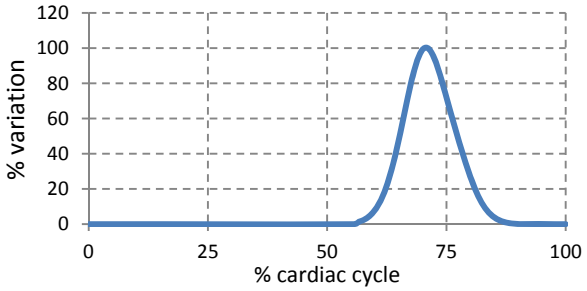

Figure 2. Percentage variation of material parameters during cardiac cycle.

Table 1. Diastolic and maximum systolic material parameters values.

Parameter	Diastolic value	Systolic maximum value
C_{10}	0.2 kPa	6 kPa
k_1	0.51 kPa	100 kPa
k_2	1.6	16

2.3. Boundary conditions

To perform the simulations, suitable boundary conditions must be applied to the ventricular model.

In particular, the boundary conditions for the simulation of an entire cardiac cycle include: kinematic constraints to avoid rigid motions of the structure; the implementation of a pre-load circuit; the implementation of an after-load circuit.

About the kinematic boundary conditions, to constrain the model, all the displacements of the ventricular base were prevented.

The ventricular pre-load and after-load circuits are shown in Fig.3.

The pre-load circuit consist of a resistance placed between a single node, representing the left atrium (LA), and the ventricle. The diastolic phase is simulated by applying a constant non zero pressure at the atrium.

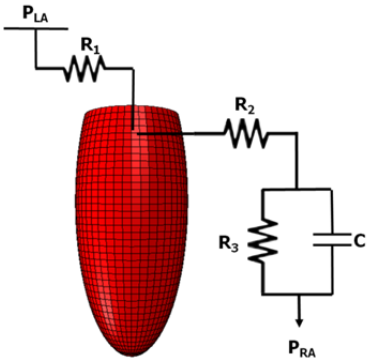

Figure 3. Ventricular pre-load and after-load circuits (P_{LA}=left atrium pressure; R_1=0.2 mmHg/(l/min); R_2=0.325 mmHg/(l/min); R_3=22.72 mmHg/(l/min); C=8*10-5 l/mmHg, P_{RA}=right atrium pressure).

At the beginning of the first cycle the ventricular pressure is zero, thus the ventricle is filled by a volume secondary to the pressure difference between the atrium node and the ventricular cavity. This inflow volume is dependent on the (pressure difference)-(flow) relationship shown in Fig.4, which results in a linear resistance of 0.2 mmHg/(l/min) during the ventricular filling phase.

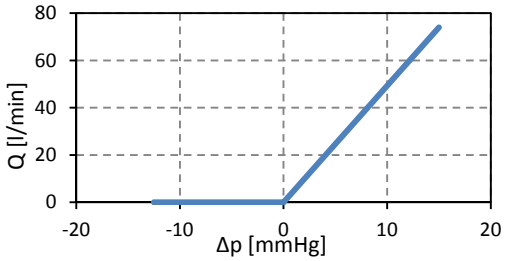

Figure 4. Pressure difference (Δp)-flow (Q) relationship set in pre-load circuit.

Instead, the after-load circuit was simulated by an RCR model, implemented secondary to a previous work [10]. Briefly, the circuit resistances were set, similarly to the pre-load circuit, by imposing a linear Δp-Q relationship between the ventricle and a node representing the aorta (R_2=0.325 mmHg/(l/min)) and between the latter and another node which represents the right atrium (RA) at a pressure of 5 mmHg (R_3=22.72 mmHg/(l/min)). Also in this circuit, backward flows were prevented.

Since a hydraulic compliance is not implemented in ABAQUS, a sphere composed of shell elements was designed to guarantee a compliant element which can simulate the systemic circulation compliance (C=8*10-5 l/mmHg).

3. Results

The results of the simulations are shown in terms of ventricular pressure-volume (P-V) relationship. To reach

the steady state of the system, a minimum of three cardiac cycles were simulated, for both models.

The application of a left atrial pressure of 4 mmHg highlights significant differences in the behaviour of the trabeculated and smooth models (Fig.5).

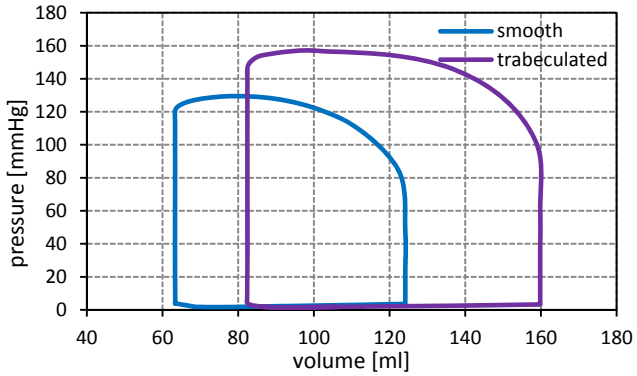

Figure 5. P-V loops of the smooth and the trabeculated model at a left atrial pressure of 4 mmHg.

In the presence of the trabecular mass, the telesystolic volume increases, as well as the telediastolic volume, compared to the smooth model; as a consequence, the systolic pressure peak is higher for the trabeculated model (157 mmHg versus 129 mmHg), as well as the stroke volume, which is 61 ml and 78 ml for the smooth and the trabeculated model respectively.

Furthermore, if the trabecular mass is present, a lower atrial pressure (-25%) is required to obtain the same stroke volume as the smooth case (Fig.6).

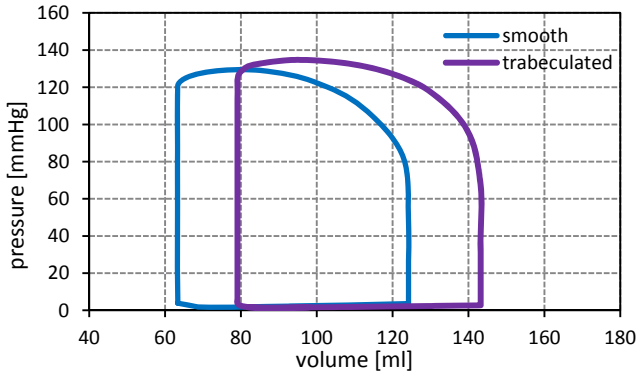

Figure 6. P-V loops imposing an atrial pressure of 4 mmHg for the smooth model and 3 mmHg for the trabeculated model.

4. Conclusions

In this work, a finite element model of the left ventricle was developed to investigate the influence of cardiac trabeculae on heart performances.

The objective was to compare the ventricle behaviour in the presence or not of the trabecular mass on the inner surface of the ventricle; for this purpose, two different models were implemented.

The simulations results suggest a significant role of cardiac trabeculae on ventricular behavior: during the diastole, the trabecular mass contributes to the ventricular filling, with an increase in both telesystolic and telediastolic volumes. As a consequence, accordingly to the Frank-Starling law, an higher stroke volume (+21.8%) is ejected by the ventricle during the systolic phase.

From another point of view, since the ventricular compliance is higher if the trabeculae are present, a lower atrial pressure (-25%) would be needed to obtain the same stroke volume.

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