CONSTRUCTION OF A FINITE ELEMENT MODEL OF THE HUMAN VENTRICLES TAKING INTO ACCOUNT THE FIBER ORIENTATION PATTERN

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INTRODUCTION

An important aspect of the human heart is related to the architecture of the ventricular muscle fibers. Unfortunately, our possibilities for determining the fiber orientation in the myocardium under *in vivo* conditions are very limited. Experimental measurements, in particular MRI imaging and tagging methods, provide mostly global information about the motion of the heart during contraction. Additionally, there is no complete theory about the dynamics of functional units of actively contracting muscle fibers including the interstitial hydraulics.

Mathematical models of the human heart are intended to facilitate an in-depth understanding of cardiodynamics, particularly as it relates to the left ventricle. Yet, the spatial resolution of image tagging patterns is quite limited. Thus mathematical models necessarily rely on extrapolations to fiber dimensions.

In most published heart models the muscle fibers are assumed to be parallel to the endocardial and epicardial surfaces, respectively, and the active stresses are directed along the fibers [1-5]. These assumptions may deviate in part considerably from the reality, in particular with regard to pathological cases.

In this work we attempt to construct a finite-element model to approximate the three dimensional contraction of the left ventricle. We include the anisotropy associated with normal or pathologic fiber structures of the myocardium as well as describe the active fiber contraction processes. The results of the simulation are assessed by comparison with results of MRI imaging and tagging. Ultimately, the model is expected to allow the derivation of information with respect to the distribution of fiber stresses in the ventricular wall. For this purpose, it is however necessary to "invert" the modeling process, i.e., to extrapolate stresses from deformations rather than determining deformations from assumed fiber contraction behavior. The question therefore arises to what extent forces and associated fiber patterns can be inferred by comparing simulation results with a known deformation pattern taken from MRI measurements.

METHODS

The first step in constructing the finite-element model was to define the geometry of the human heart. Two geometric models were created for this purpose, a closed surface for the left ventricle and another for the left and right ventricles together. The endocardial and epicardial surfaces were reconstructed by digitizing surface points from a human *post mortem* heart. Approximately 1,900 points on the endocardial surface and 4,650 points on the epicardial surface of the left ventricle were digitized. For the right ventricle, in turn, about 3,200 points on the endocardial surface and 3,400 points on the epicardial surface were digitized.

A finite element mesh was created from the three dimensional surfaces. A mesh with more than 47,000 elements was necessary for the left ventricle in order to include the desired level of anatomical detail, in particular, as it relates to the fiber orientation field (see below). For the second model, the mesh had around 104,000 elements. The elements were eight-node, isoparametric, arbitrary hexahedral (Fig.1).

To obtain representative fiber architectures of the human heart, a fiber peeling method was applied to selected hearts [6]. These data contained the coordinates of several thousand myocardial fibers, whereby each fiber, in turn, was represented by a number of points. We used these points to reconstruct the curves that document the fiber trajectories in the myocardium. Due to the method of the digitization, however, the measured fibers were not evenly distributed throughout the myocardium and the pattern had to be completed accordingly.

For our finite element model it was necessary to determine the spatial fiber direction in each element. To achieve this goal, an algorithm was developed such that for any arbitrary mesh with sufficiently small elements a well-defined fiber orientation could be determined. The algorithm determined the tangent of the fiber trajectories at the central point of each element. With the peeling method, approximately 2700 left ventricle fibers and 4500 ventricular fibers for the combined left and right ventricles in the selected heart were detected, respectively (Fig.2).

Another feature of the software was the building of a 2D-representation of layers with defined thickness and viewpoints from an arbitrary perspective within the myocardium, which is needed to verify the fiber direction field.

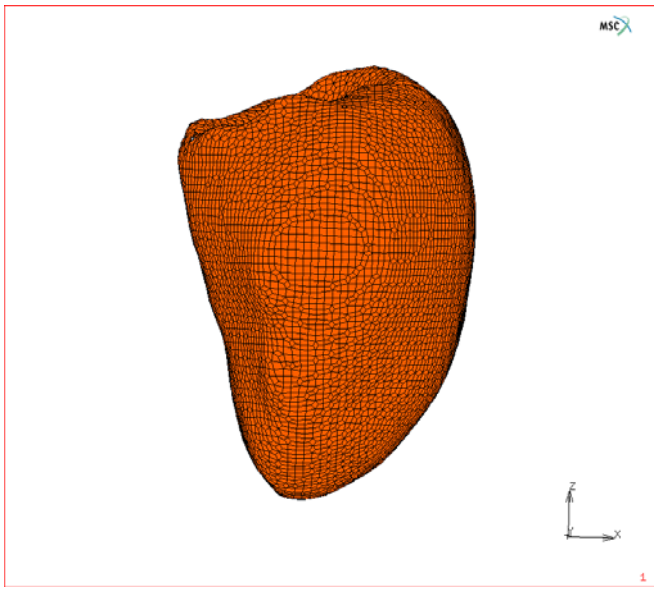

Figure 1. Mesh of a typical left ventricle

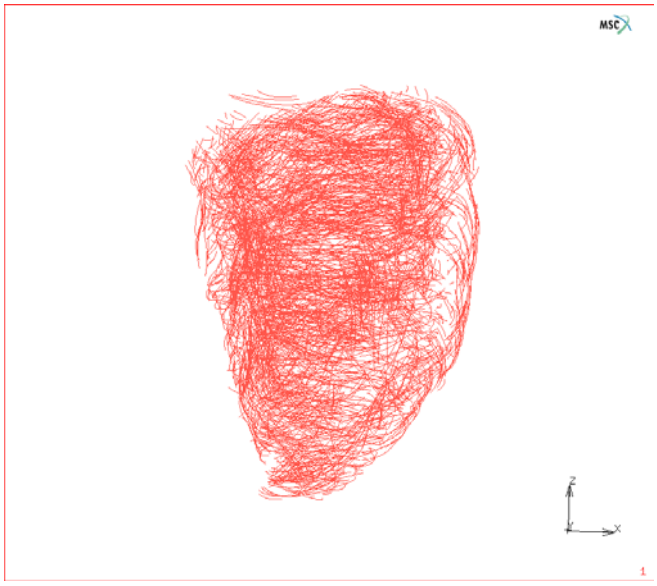

Figure 2. Fiber trajectories of a typical left ventricle

A local rectangular Cartesian coordinate system was defined for each element and the transformation matrix to the global rectangular Cartesian coordinate system was determined. Contraction was modeled by defining the second Piola-Kirchhoff stress tensor with respect to the global coordinate system in the unloaded body. This tensor consisted of two contributions, viz., the components of the passive stress derived from a strain energy function describing the passive myocardial material and the components of the active stresses due to fiber contraction.

The procedure consisted of defining at the beginning of each increment, first, the active stresses in the local coordinate system for each element and, second, calculating the aforementioned corresponding components in the global coordinate system utilizing the coordinate transformation. These components were then added to the second Piola–Kirchhoff stress tensor. Thus, this tensor was updated accordingly at each increment. Physiological curves were used for the blood pressure in the left and right ventricle and the equilibrium configuration was determined at each integration step. It is thereby implied that inertia forces can be disregarded throughout the heart cycle.

RESULTS AND DISCUSSION

Three-dimensional geometric models of the left ventricle and left and right ventricles, respectively, were constructed and finite element models for both geometries were implemented. Algorithms were developed which allowed one to study the fiber structure, to implement it into the finite element model and to provide the possibility to cut graphically an arbitrary layer and verify the fiber orientation in different areas of the myocardium.

Three-dimensional representations of the whole fiber structure of the left and right ventricles were used to determine the tangents of the fiber trajectories in each element and as such create a vector direction field for the entire myocardium. The contraction of the heart was simulated for a model of the left ventricle alone as well as for a model of both the left and right ventricles together.

Results were then compared with high-resolution MRI images. In particular, the longitudinal shortening of the heart and the transversal wall thickening were considered. Although both the particular fiber field implemented in the FE model as well as the MR measurement (performed on a healthy volunteer) exhibited individual variations and therefore local differences, general characteristics of the contraction process could be verified. The fact that the wall thickening pattern shows local inhomogeneities even under healthy conditions indicates that wall thickening qualifies as a sensitive parameter to study cardiodynamics.

Work is continued in view of modeling pathologic situations such as myocardial infarction or hypertrophy by changing the local fiber structure and contraction behavior.

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