Mechanics of the human ventricles

Jana van Gerwen

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Supervisors:

Martyn Nash
(Bioengineering Institute, The University of Auckland)
Vicky Wang
(Bioengineering Institute, The University of Auckland)
Peter Bovendeerd
(Biomedical Engineering, Technical University of Eindhoven)
Abstract

To investigate the cardiomyopathy in, for example, myocardial infarction, it is important to understand ventricular mechanics during the cardiac cycle. 3D finite element models can be used to simulate the whole-organ response, and also to predict quantities that cannot be measured directly, such as mechanical stress and work in the beating heart muscle.

A finite element representation of both the left and right human ventricles was created to realistically represent the ventricular anatomy. A reference finite element model was fitted to a surface data set of the human ventricles derived from DT MR images. Fibre directions were imbedded into the model in order to represent the material anisotropy of the tissue. The model was suitable for simulating the passive inflation phase and isovolumic contraction phase of the cardiac cycle. In order to make the model useful to investigate a cardiomyopathy, it needs to be further expanded and improved.
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Chapter 1

Introduction

Heart failure is the leading cause of death in the industrialized world. Myocardial infarction is one of the most common pathologies. The onset of myocardial infarction starts a remodelling response that changes the mechanical properties of the heart wall. Knowledge of this response is essential for determining the optimal treatment. Anatomically realistic computer models can be used to simulate this behavior and predict quantities that cannot be measured, such as mechanical stress and work in the beating heart muscle. So far, the models used to study such cardiomyopathies often only contain the left ventricle [15]. To accurately investigate cardiac behavior, the right ventricle also needs to be taken into account. A biventricle model could also be used to investigate other cardiomyopathies as infarction, such as ventricular fibrillation [6]. Biventricle models exist, but are often based on porcine or canine data [14], [11]. In this study, a geometrical and mathematical model of both ventricles based on human DT MRI data was developed.

The human ventricles have a complex asymmetric shape and anisotropic fibrous structure. To determine the stress and strain fields, it is therefore essential to use three-dimensional finite element analysis. A finite element model of both the left and right ventricle, that realistically represents the ventricular anatomy was created. To achieve this, a reference model was fitted to a surface data set of the human ventricles derived from DT MR images. Fibre directions were embedded into the model in order to represent the material anisotropy of the tissue. Since material deformations during the cardiac cycle are large, large deformation elasticity theory was used. The resulting model can be used to simulate different phases of the cardiac cycle. In this study, the model was used to describe the diastolic (inflation) phase and the isovolumic contraction phase.

Chapter 2 describes the extraction of the surface and fibre data of the human ventricles. The principles and results of the finite element model are given in chapter 3. The simulations of mechanics are described in chapter
4. In chapter 5 the whole study is discussed and concluded.
Chapter 2

The surface and fibre data of the human ventricles

To realistically describe the geometry of the heart a finite element model was fitted to a surface data set. This data set was derived from DT MR images and consists of the epicardial surface and the endocardial surfaces of both the left and right ventricles. After this fitting procedure fibre orientation data can be embedded into the model. Although a fibre data set of the same heart was available it was not used due to time limitations. Instead a fibre field based on fibre orientations derived from literature was used.

2.1 DT MRI

The data set that was used in this study was obtained with diffusion tensor magnetic resonance imaging (DT MRI). This technique combines NMR imaging principles with principles that encode molecular diffusion effects in the NMR signal by using bipolar magnetic field gradient pulses. Diffusion of water molecules has been shown to be faster in the direction of the fibres than in the perpendicular directions [4]. With plain diffusion MRI, diffusion is described by the diffusion coefficient $D$, which is a scalar parameter. However, in the presence of anisotropy, this is no longer sufficient and diffusion requires a tensor $\mathbf{D}$ to describe molecular mobility along each direction and the correlation between these directions. So, unlike plain diffusion MRI, DT MRI can characterize the anisotropy of the diffusion and therefore the directions of the fibres. For fibre orientation mapping, the eigen-vectors of the diffusion tensor, associated with the largest eigen-diffusivity is taken as the direction of the fibres. DT MRI is used most often for fibre tracking in the brain. This technique also has a tremendous potential of providing data on heart fibres, but it remains technically very challenging to perform in vivo measurements due to heart motion and blood perfusion. However, DT MRI can be used ex vivo to measure fibre orientations in the myocardium.
2.2 The surface data set of the human ventricles

The human DT MRI data set was provided by the National Institute of Health [13] and consisted of 134 slices with a thickness of 1.0 mm. During the ex vivo fast spin echo measurements, the heart was placed in a container filled with Fomblin and the long axis of the heart was aligned with the z-axis of the scanner. The resolution of the gray scale MR images of the slices was 256 by 256 pixels (0.43 by 0.43 mm). For each slice the epicardial surface, left ventricle endocardial surface and right ventricle endocardial surface were manually digitized and the resulting data sets contained 3940, 3927 and 2114 data points, respectively. During the digitization the trabeculae and papillary muscles were neglected. This was done to prevent the problems that occurred in the past with models that were based on data that included these muscles which made the anterior and posterior walls artificially thick compared to the left ventricle free wall [9]. Due to poor image resolution in some regions, the data points in those regions were chosen such that the surfaces were relatively smooth. Therefore the data points may not always accurately describe the true surfaces. Afterwards, the data points that represent the apexes were extrapolated. Around the right ventricle outflow tract, no data was created as can be seen in figure 2.2, leaving a gap in the data set. The final data set is displayed in figure 2.1. Prior to fitting the model to the surface data, the data had to be transformed into the cardiac coordinate system. In this system, the x-axis is pointing from base to apex, the y-axis from the left to the right ventricle and the z-axis is...
2.3 The fibre orientation field

The fibre field that was embedded into the fitted model was derived from experimental canine data found in the literature [10]. Fibre orientations can be described as a function of the fibre angle, imbrication angle and sheet angle. For simplicity only the fibre angles were implemented, since they provide the largest contribution to the fibre orientation. The fibre angle was defined as the angle between the fibre vector and the circumferential direction (short-axis plane) of the model. In the model, the transmural variation of fibre angles was similar for the left ventricle and right ventricle free walls, with the orientation changing from 90° at the endocardium to -60° at the epicardium. In the septum, however, the transmural variation changes from -90° at the left ventricular endocardium to 90° at the right ventricular endocardium. At the ventricular apex and the bottom of the right ventricle, the fibre angle was set to 0°.
Chapter 3

An anatomically realistic finite element model of the human ventricles

To build a model of the complex geometry of the ventricles and the non-linear behavior of the material, the domain was discretised into a number of tricubic Hermite elements. Parameters of interest defined at the finite element nodes can be continuously interpolated. The numerical methods developed for creating the finite element model and solving mechanical problems were implemented in CMISS. CMISS (Continuum Mechanics, Image analysis, Signal processing and System identification [2]) is a mathematical modeling environment developed by researchers at the Auckland Bioengineering Institute. It can be used as a modelling tool for solving (nonlinear, time dependent) partial differential equations over complex domains. CMISS is a combination of two software packages: cm and cmgui. Cm is written in Fortran and used for computational modelling and cmgui is a graphical user interface and can be used for visualization and manipulation of a finite element model. In this study, cmgui was also used to extrapolate the apical data points. Zinc is a browser extension that embeds the cmgui visualization engine and was used in this study to digitize MRI images as described in chapter 2.

In the elements the local normalized coordinates $\xi_1$, $\xi_2$ and $\xi_3$ were used, which represent the circumferential, longitudinal and transmural directions, respectively. To create the reference model, an existing model [14] was used as a starting point. The nodal positions of the reference model were placed such that they approximate the human surface data set of both ventricles, described in chapter 2. The global coordinate system is the cardiac coordinate system.

To create an anatomically realistic model of the human ventricles, the reference model was fitted to the surface data. In this chapter, the basic
principles behind the reference (unfitted) model (that can be seen in figure 3.1) and the fitting procedure are explained and the results of the fitting procedure are given.

3.1 Cubic Hermite elements

Compared to linear Lagrange elements, cubic Hermite elements can capture the shape of an object more accurately with fewer number of elements, since they also preserve continuity of first derivatives across element boundaries (i.e. $C^1$ continuity). This continuity is also advantageous for the mechanical simulations, described later, since $C^1$ continuity of the geometry ensures that the strain distribution, and therefore the stress distribution, are spatially continuous. Trilinear Lagrange interpolation provides an adequate resolution and continuity of the hydrostatic pressure field. To use this interpolation, the interpolation scheme for the geometric solution fields ($x$, $y$ and $z$) should always be of higher order (e.g. tricubic) to ensure continuous stress distribution. In cubic Hermite elements, in addition to nodal values, the derivatives of the coordinates with respect to the local coordinate $\xi$ are used to describe a shape. This means that cubic Hermite elements have many more degrees of freedom (DOFs) per node than linear elements. For 1D elements, this makes the interpolation function to be as described in
The four 1D cubic Hermite basis functions that define the shape of the interpolation of $x$ are given in equation 3.2. In this equation and equation 3.1 $\psi_i^j(\xi)$ denotes the shape function for node $i$ and derivative $j$.

Because $\frac{dx}{d\xi}$, defined at node $n$ depends upon the local $\xi$-coordinate, this derivative is often different in two adjacent elements. Therefore, a physical arc-length derivative, $\frac{dx}{ds}$, and equation 3.3 were used, to determine $\frac{dx}{d\xi}$ [5].

$$\frac{dx}{d\xi}|_n = \left(\frac{dx}{ds}\right)_{\Delta(n,e)} \cdot \left(\frac{ds}{d\xi}\right)_e$$

(3.3)

Here $\Delta(n,e)$ is the global node number of local node $n$ in element $e$ and $(\frac{ds}{d\xi})_e$ is an element scale factor, denoted by $S_e$. Thus $\frac{dx}{d\xi}$ is constrained to be continuous across element boundaries rather than $\frac{dx}{ds}$. One more condition must be placed on $\xi$ to ensure we have arc-length derivatives: the arc-length derivative vector has to be normalized. Using this, the unknown average scale factor can be calculated from the scale factor, as displayed in equation 3.4.

$$\frac{\partial s}{\partial \xi} = \int_0^1 \frac{\partial s}{\partial \xi} \cdot d\xi = S_e$$

(3.4)

To ensure $C^1$ continuity, the scale factor at a node in one element should be the same at that node in an adjacent element. It is desirable to have uniform spacing of $\xi$ with respect to arc-length (for example, not biasing the Gaussian quadrature scheme to one end of the element). To achieve this uniform spacing, the nodal scale factor can be set to be the average of the two arc-lengths on either side of the node. The set of mesh parameters $u$, for cubic Hermite interpolation hence contains the set of nodal positions, the set of nodal arc-length derivatives and the set of scale factors.

For 3D cubic Hermite volume elements, or tricubic Hermite elements, there are 8 DOF per node for every direction: $x$, $\frac{\partial x}{\partial \xi_1}$, $\frac{\partial x}{\partial \xi_2}$, $\frac{\partial x}{\partial \xi_3}$, $\frac{\partial^2 x}{\partial \xi_1 \partial \xi_2}$, $\frac{\partial^2 x}{\partial \xi_1 \partial \xi_3}$, $\frac{\partial^2 x}{\partial \xi_2 \partial \xi_3}$ and $\frac{\partial^3 x}{\partial \xi_1 \partial \xi_2 \partial \xi_3}$. This means that the total number of DOF per node is 24.

### 3.2 Geometric fitting

The reference model and the data are displayed in figure 3.2. The fitting procedure was a surface fitting, the left ventricular endocardial face of the
Figure 3.2: The reference model plotted with the human surface data set, with the right ventricle endocardial surface displayed in blue, the left ventricle endocardial surface displayed in green and the epicardial surface displayed in yellow.

reference model has been fitted to the left ventricular endocardial surface data. The right ventricular endocardial face and the epicardial face, have been fitted to the right ventricular endocardial surface data and the epicardial surface data respectively. Since this was a surface fitting, the parameters associated with the transmural ($\xi_3$) direction were not altered by the fit.

During the fitting procedure, the distance between the data points and the surface of the mesh was minimized [5]. For every data point ($z_d$), the orthogonal projection ($z_p$) on the mesh is calculated. In a 2D case, $z_d$ can be interpolated from a given local element coordinate $\xi_d$. The Euclidean distance between $z_d$ and $z_p$, the projection error, can be calculated for each data point. From this, a fitting objective function (OF), i.e. the sum-of-squares as displayed in equation 3.5 can be determined.

$$OF = \sum_{n=1,D} \| z(\xi_d) - z_d \|^2$$  (3.5)

The objective function needs to be minimized with respect to every DOF of the mesh, i.e. optimum values of the DOFs need to be determined. If the scale factors are kept constant during the fit, this will result in a linear system $Ku = f$, where $K$ is the global stiffness matrix and $f$ the zero vector. This system can be solved to yield the nodal parameters ($u$) which minimize the projection errors in the mesh. Unfortunately with cubic Hermite elements, this linear system is not ideal, since it is important to maintain the arc-length derivatives and average arc-length scaling during the fit. Because both the value of the arc-length for the element and the
relationship between the derivatives in the various spatial directions depend upon the mesh parameters in a non-linear fashion, the only way to ensure arc-length derivatives are maintained during fitting is to use a non-linear fitting procedure. This can be achieved by keeping the scale factors constant during the fit and update them afterwards (based on the new mesh) to be average arc-length. This process can be repeated iteratively until the desired fit has been achieved. Unfortunately, due to the complex geometry used in this study, this was not possible. The data point projections were updated after each iteration. To assess the effectiveness of a fit, the root mean square (RMS) error (as in equation 3.6, with \( e \) as the Euclidean distance and \( N \) as the number of data points) before and after the fit can be compared.

\[
RMS_{error} = \sqrt{\frac{\sum_{n=1}^{N} e_n^2}{N}} \quad (3.6)
\]

When the data is insufficient or scattered, the fitted structure may have an undesirable shape. Therefore, Sobelov smoothing constraints can be introduced. These smoothing constraints are incorporated as penalty functions in the fitting objective function. Basically these penalty functions ‘penalize’ for excessive curvature of arcs, arc-lengths and surface areas and for excessive volume of the elements. The resulting objective function is:

\[
OF = \sum_{n=1,D} \| z(\xi_d) - z_d \|^2 \\
+ \int_{\Omega} \left\{ \alpha(\| \frac{\partial u}{\partial \xi_1} \|^2 + \| \frac{\partial u}{\partial \xi_2} \|^2) + \beta(\| \frac{\partial^2 u}{\partial \xi_1 \partial \xi_2} \|^2 + 2\| \frac{\partial^2 u}{\partial \xi_1^2} \|^2 + \| \frac{\partial^2 u}{\partial \xi_2^2} \|^2) \right\} d\xi \quad (3.7)
\]

\( \alpha \) is the smoothing factor that controls the arc-lengths, while \( \beta \) controls the surface and the arc curvatures. Both smoothing factors were determined arbitrarily and the best values were determined by evaluating different fits.

### 3.3 Mapped and fixed degrees of freedom during the fitting procedure

In a collapsed element one or more nodes are placed at the same nodal position as another node of this element. As a result, a local coordinate is nonexistent or collapsed. The collapsed nodes at the apex and the nodes in the elements at the right ventricle insertion had to be mapped together during the fitting procedures and simulations to prevent holes from opening up during these procedures. A mapping is done to ensure that the DOFs of the different versions of a node stay the same when this is desired. The mapping that was used for the surface fitting procedure was also used for the simulations of mechanics. If it is wished to maintain a DOF, that DOF can be fixed so it will not change.

It was desired for the fitted geometry to have such a flat basal plane as the unfitted geometry has. This was achieved by fixing the x-coordinate and
the $\frac{\partial x}{\partial \xi_1}$ derivative. As can be seen from figure 3.2 one face near the base is surrounded by only a couple of data points due to the removal of the right ventricle outflow tract. To make sure that the DOFs of the node adjacent to this face behave well, the y and z-coordinates were also fixed.

### 3.3.1 The collapsed apical nodes

At the ventricular apex, collapsed elements were used to describe the geometry of this region. In this model, in every apical element two or four nodes were collapsed. This means that two or four of the eight nodes in this element have the same geometric location. In this case the circumferential direction ($\xi_1$) was collapsed, as can be seen in figure 3.3. In this figure the collapsed epicardial apical and left ventricle endocardial apical nodes are displayed with two adjacent elements. The element at the bottom has two nodes that were collapsed into one epicardial apical node. Two other nodes were collapsed into the endocardial apical node. Since the $\xi_1$-direction was collapsed, the line between the two apical nodes is a transmural direction. From the epicardial apical node two different $\xi_2$-directions innate, that is, this node has two versions. To describe the apex correctly, these two $\xi_2$-directions need to have a different shape, i.e. different nodal derivative values. The element at the top has four collapsed nodes, resulting in one endocardial apical node. For this node the $\xi_1$ and $\xi_3$-directions were collapsed and there are four versions of the $\xi_2$-direction. In this model, the endocardial apical node has 11 versions, while the epicardial apical node has 9 versions. The endocardial apex has more versions, since some of these versions represent the lines that go to the nodes at the bottom of the right ventricle, as depicted in figure 3.3.
For the apical nodes, the nodal coordinates for the different versions were mapped together. Also, the line in the $\xi_3$-direction between the two collapsed apical nodes (displayed in figure 3.3) was mapped to be the same for every version of both nodes. Both nodes were placed in the right position and their nodal coordinates were fixed to stay the same during the fitting procedure. For the epicardial apical node, the $\frac{\partial x}{\partial \xi_1}$ parameter was fixed for every version, to maintain the smooth shape of the epicardial apex. The derivatives of $y$ and $z$ in the $\xi_2$-direction were left free. For the endocardial apex node, this was the case for all the global coordinates.

3.3.2 The elements at the right ventricle insertion

The nodes at the posterior and anterior insertion of the right ventricle both have a line that connects the left ventricle endocardium with the right ventricle endocardium. The nodes at the ends of these lines have multiple versions, the lines and nodes are highlighted in figure 3.4. From this figure it can be seen that three $\xi_1$-directions start from node A and D, of which two describe the endocardium of the left ventricle and one the line through the septum. Node B and C also have three versions each, since these versions need to describe the septum, the endocardial right ventricle free wall and the line that connects the endocardium of the left ventricle to the endocardium of the right ventricle.

Unfortunately the three versions of the nodes at the right and left ventricle insertion cause the derivatives to be inconsistent for the adjacent elements (i.e. $C^1$ discontinuity). Namely the lines that connect the left ventricle en-
docardium with the right ventricle endocardium, represent the $\xi_1$-direction in one element (for example element 1 in figure 3.4), while in the adjacent element they represent the $\xi_3$-direction [14]. To prevent the development of holes at the line between node A and B and between node C and D, during for example a fitting procedure, mappings need to be done. As can be seen in figure 3.1, there are four of these planes in the model, which all have these type of nodes with versions. This means that node A, B, C and D can be considered as categories of nodes, their detailed mappings can be found in Appendix A.

3.3.3 The bottom of the right ventricle

At the bottom of the right ventricle, the elements of the septum and the right ventricle free wall come together. This means that in the circumferential direction, there are two nodes between node B and C (figure 3.4). These nodes have two versions each, since the $\xi_2$-direction of one version has to describe the septum and the $\xi_2$-direction of the other version has to describe the endocardium of the right ventricle free wall. The nodal coordinates of both versions of these nodes were mapped together, as well as the derivatives in the $\xi_1$ and $\xi_3$-directions.

The four nodes in the unfitted geometry, that are at the bottom of the right ventricle were placed on or close to the corresponding data points and the nodal coordinates were fixed during the fit. This was done to enhance the shape of the right ventricle at these points. The selected data points were not included in the fit to prevent them from projecting to the wrong surfaces.

3.4 Fitting the model

The model consisted of 48 tricubic Hermite elements, 88 nodes and 2774 DOFs. During the calculation of the projections, some constraints were applied to optimize this calculation. The projections were allowed to cross the element boundaries. For the epicardial and left ventricle endocardial surface, the number of faces that the data points were first tried to project to was set to 6. For these surfaces, a grid of 6 by 6 points was formed on each face. These grid points were a starting position to begin a non-linear search for the closest point. For the left ventricle, the initial projections were used to calculate the projections after the first iteration. Due to the complicated shape of the right ventricle, only 4 faces were used to try to project the data points to. Also, it was attempted to use orthogonal projections only.

Of the epicardium and the left ventricle endocardium all data points were included in the surface fitting. During the right ventricle endocardial fitting procedure one data point could not be projected to a surface of the model. This means that for the epicardial surface 427 DOFs were fitted to
3940 data points, for the left ventricle endocardial surface 435 DOFs were fitted to 3927 data points and for the right ventricle 312 DOFs were fitted to 2113 data points.

3.5 Results

The smoothing factors that were determined for all the surfaces were $\alpha = 0.05$ and $\beta = 0.025$. Five iterations were done, because after this iteration the RMS error would no longer significantly decrease. The resulting RMS values of the surface fitting procedure can be found in table 3.1. For comparison, the data measured from the basal plane to the epicardial apex is about 90 mm.

Table 3.1: RMS values for the epicardial, left ventricle (LV) endocardial and right ventricle (RV) endocardial surfaces

<table>
<thead>
<tr>
<th>iteration</th>
<th>RMS error [mm]</th>
<th>epicardium</th>
<th>LV endocardium</th>
<th>RV endocardium</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>2.84</td>
<td>1.96</td>
<td>3.39</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>0.52</td>
<td>0.45</td>
<td>0.66</td>
<td></td>
</tr>
</tbody>
</table>

After the fitting procedure, the total volume of the myocardium was 198 ml and the cavity volumes of the left and right ventricles were 125 and 118 ml, respectively. The fitted mesh and the data it has been fitted to are illustrated in figure 3.5. Figures 3.6, 3.7 and 3.8 show the fibre orientation distributions, which were embedded into the model, on the left ventricular endocardium, the right ventricular endocardium and the epicardial surface respectively. The transmural fibre orientation distributions can be seen in figure 3.9.

3.6 Discussion

The fibre angle is defined with respect to the circumferential direction. The versions of the nodes at the right and left ventricle insertion cause the fibre field to be discontinuous, since the line that connects these nodes is a $\xi_1$-direction in one element while it is a $\xi_3$-direction in the adjacent element, as displayed in figure 3.4. This discontinuity can be seen in figure 3.10. In the future, this problem could be solved by imposing a imbrication angle for the versions at the right and left ventricle insertion.
Figure 3.5: The fitted model plotted with the human surface data set, with the right ventricle endocardial surface displayed in blue, the left ventricle endocardial surface displayed in green and the epicardial surface displayed in yellow.

Figure 3.6: The fibre distributions on the septal wall (left) and the left ventricular free wall (right)
Figure 3.7: The fibre distributions on the septal wall (left) and the right ventricular free wall (right)

Figure 3.8: The fibre distributions on the epicardium: anterior view (left) and posterior view (right)
Figure 3.9: The transmural fibre distributions in the right ventricle free wall and septum (left) and in the left ventricle free wall (right)

Figure 3.10: The fibre field of the basal plane, with red indicating a 90° fibre angle, green a 60° fibre angle and blue a 0° fibre angle
Chapter 4

Simulations of mechanics of the diastolic and isovolumic contraction phase

The cardiac cycle can be divided into the four phases that are depicted for the left ventricle in figure 4.1. The first phase is the diastolic (passive filling) phase during which the ventricles relax. When the pressure in the ventricles is lower than the pressures in the atria, the atrioventricular valves open and the blood flows from the atria into the ventricles, expanding the ventricles. The last 20% of the filling is accomplished when the atria contract. Ventricular systole begins at the apex of the heart and the blood is pushed upwards to the atrioventricular valves, forcing them to close. With all valves closed, the ventricles continue to contract and the cavity pressure increases. This phase is called the isovolumic contraction. The ventricles contract further and generate enough pressure to open the aortic and pulmonary semilunar valves, pushing the blood into the arteries, during the ejection phase. At the end of each ventricular contraction, the ventricles begin to relax. This causes the ventricular pressure to decrease and when this pressure falls below the pressures in the arteries, a backflow of blood closes the semilunar valves. The ventricles become a sealed chamber again and relax further during this isovolumic relaxation. This completes the cardiac cycle.

To simulate the different phases of the cardiac cycle, a finite deformation elasticity problem in a Lagrangian framework can be formulated. The equations that govern the motion of the deformable material can be derived from four relations: kinematic relations, stress equilibrium, constitutive relations and boundary conditions. In this chapter the constitutive equation that was used for the simulations in this study is given. It is explained what the displacements boundary conditions were and how the system of equations was solved. Next, it is explained how the simulations of the different phases of the cardiac cycle were set up and the results of the simulations are given
Figure 4.1: The cardiac cycle
and discussed.

4.1 Constitutive relations

Constitutive equations characterize individual materials and their response to external loads. The relation between deformation and stress in the incompressible material is given by the following constitutive relation:

\[ T_{MN} = \frac{\partial W}{\partial E_{MN}} + J \frac{\partial X}{\partial x_i} p^i_j \frac{\partial X}{\partial x_j} \] (4.1)

with strain energy density function \( W \) as a function of the Lagrangian Green’s strain tensor \( E \). The second Piola-Kirchhoff stress tensor is used rather than the Cauchy stress tensor, because it refers all stresses back to a known reference state instead of the unknown deformed state. A transversely isotropic strain energy density function as a function of \( E \), determined by Guccione et al [8], was chosen to model the passive myocardium:

\[ W = \frac{1}{2} C_1 (e^Q), \]

where \( Q = b_1 E_{ff}^2 + b_2 (E_{cc}^2 + E_{rr}^2 + 2E_{cr}E_{rc}) + 2b_3 (E_{cf}E_{cf} + E_{rf}E_{rf}) \) (4.2)

The strain components \( E_{ij} \) refer to a system of local fibre, cross-fibre and radial coordinates (f, c and r respectively). The material parameters of this strain energy function were identified for the canine midwall by Omens et al [12] and are depicted in table 4.1. These parameters were assumed to be homogeneous throughout the whole myocardium.

Table 4.1: Material parameters of the exponential transversely isotropic strain energy function of equation 4.2

<table>
<thead>
<tr>
<th>( C_1 [\text{kPa}] )</th>
<th>( b_1 )</th>
<th>( b_2 )</th>
<th>( b_3 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.2</td>
<td>26.7</td>
<td>2.0</td>
<td>14.7</td>
</tr>
</tbody>
</table>

4.2 Displacement boundary conditions

All nodes of the basal plane and their in-plane first derivatives and cross derivatives were fixed to prevent rigid body rotation. To make convergence possible all first derivatives and cross derivatives had to be fixed, except for the first derivative in the longitudinal direction of the endocardial apical node. The same was done for the epicardial apical node. This makes the number of DOFs 2050.
4.3 Solving the system of nonlinear finite element equations

There are four solution fields to solve the system of governing equations described above: the geometric coordinates x, y and z and the hydrostatic pressure p. The equations are rearranged into a nonlinear set of residuals, which was solved with respect to the set of solution variables using the Newton-Raphson method. This set consists of the positions (or displacements) and their arc-length derivatives in each of the coordinate directions at each global node of the finite element mesh. Because the myocardium is an incompressible material, additional constraints arise. The resulting set of linear equations was solved with either the generalized minimum residual method or the LU decomposition method.

The initial solution for the finite element equations for the deformation is chosen to be the undeformed mesh. This makes convergence for small loads likely. For the larger loads, used in this study, convergence is more likely when the applied load is split up into incremental load steps and are applied sequentially. The final solution from the previous load step is used as the initial solution for the current load step. Convergence is achieved when both the ratio of unconstrained to constrained residuals and the sum of the solution vector increments for the current Newton iteration are less than a prescribed error tolerance. Constrained residual equations are associated with DOFs for which the boundary conditions have been fixed, which means that these equations are removed from the problem. Unconstrained residuals are associated with the solution variables which are to be determined.

4.4 Simulating the different phases of the cardiac cycle

It was assumed that the surface data of both ventricles was obtained from a heart that was in zero load state, meaning that there was zero pressures in the ventricles of the undeformed mesh. Due to time limitations, only the passive inflation phase and the isovolumic contraction phase were simulated.

4.4.1 Passive inflation

To simulate passive diastolic filling, the anatomically realistic biventricle model was inflated to left ventricle and right ventricle end-diastolic pressures of 1 kPa and 0.2 kPa respectively. The load for both ventricles was split up into 10 incremental steps of 10% of the final load each to achieve convergence. The volume during diastasis and the end diastolic volume of a typical heart are about 90 and 135 ml respectively. Since this model has a diastolic volume of 125 ml, it was tried to achieve a similar relative increase in volume during
the inflation. To achieve convergence, the $C_1$ parameter of the strain energy function (equation 4.2, table 4.1) had to be increased to 1.3 kPa.

### 4.4.2 Isovolumic contraction

During the isovolumic contraction phase the myocardium is activated and calcium is enabled to enter the myocardial cells, leading to contraction. This process is simulated by increasing a non-dimensional parameter which represents the level of activation. The relationship between this parameter ($C_{a_{\text{actn}}}$) and the active tension is given in equation 4.3 [9].

$$T_{\text{active}}(\lambda, C_{a_{\text{actn}}}) = \frac{(C_{a_{\text{actn}}} \cdot [Ca^{2+}]_{\text{max}})^h}{(C_{a_{\text{actn}}} \cdot [Ca^{2+}]_{\text{max}})^h + (c_{50})^h} \cdot T_{\text{ref}} \cdot [1 + \beta(\lambda - 1)] \quad (4.3)$$

where

- $\lambda$ is the extension ratio,
- $[Ca^{2+}]_{\text{max}} = 1$ mM is the intracellular calcium concentration for maximal activation,
- $c_{50} = 0.5$ mM is the intracellular calcium concentration at which the isometric tension is 50% of its maximum,
- $h = 3$ is the Hill coefficient, determining the shape of the saturation curve,
- $T_{\text{ref}} = 100$ kPa is the isometric, actively developed tension at $\lambda = 1$ and saturating intracellular calcium.

When the activation parameter is turned on, it was assumed that all myocardial cells were activated simultaneously. The tensile stress generated by the contracting cells must be calculated and added to the passive three-dimensional stress tensor of equation 4.1. It was assumed that the muscle fibres only generate force in the direction of their current longitudinal axes and therefore only one term needs to be added to the passive Cauchy stress tensor. The total second Piola-Kirchhoff stress tensor now becomes:

$$T_{MN} = \frac{\partial W}{\partial E_{MN}} + J \frac{\partial X_M}{\partial x_i} \rho \delta_{ij} \frac{\partial X_N}{\partial x_j} + J \frac{\partial X_M}{\partial x_1} \frac{\partial X_N}{\partial x_1} \cdot T_{\text{active}} \quad (4.4)$$

with $T_{\text{active}}$ as in equation 4.3. To simulate this isovolumic contraction, the activation parameter is incrementally increased. For each level of activation the ventricular cavity pressures were determined such that they balanced the increasing fibre stress to keep the cavity volume constant. A mesh of both the left and right ventricular cavity was created to determine the volume after each activation increment. If the volume of the left ventricle had changed, the pressure in that ventricle was increased until the end diastolic
volume was reached again. The pressure of the right ventricle was always set to be 20% of the pressure in the left ventricle. The end of the isovolumic contraction was defined to occur when the left ventricular cavity pressure reached 11 kPa.

4.5 Results

The simulation of the passive inflation took about 2 hours, the isovolumic contraction simulation took about 1 day. These long simulation times are partially caused by the fact that the model was solved for many small steps with full convergence at each step to create a movie. The simulation could be much quicker when the model was only solved for the end of both phases. The graphical results of the simulation can be seen in a short movie [7]. In figures 4.2, 4.3 and 4.4 the model can be seen in the initial state and after the inflation and isovolumic contraction. During passive inflation, the left ventricular cavity increased from 125 to 174 ml and the right ventricular cavity slightly increased from 118 to 119 ml. The relationship between the increasing volume and the pressure can be found in figure 4.5. During the inflation the apex to base length increased from 90 mm to about 98 mm, during the isovolumic contraction this axial length decreased to approximately 93 mm.

The transmural strain and shear strain were determined at the end of inflation and at the end of the isovolumic contraction phase at five locations around the equator of the ventricles, these distributions can be seen in figures 4.6 and 4.7. It can be seen that at the end of the isovolumic contraction phase, the strains vary more widely. The amplitude and transmural gradient of the fibre strain ($E_{ff}$) in the anterior left ventricle free wall, at the end of the passive inflation phase is consistent with experimental data [11]. It can be seen that the radial strain ($E_{rr}$) inversely correlates with the fibre strain. If the wall thins, which is expressed as a negative radial strain, the fibres lengthen in the fibre direction, which is generally the case. The predictions of the model at the end of inflation show a relatively uniform distribution through the wall, which means that the fibres are equally contractile through the wall. The shear strain components are low compared with the normal strain components, which is consistent with experimental data [1], [11].

4.6 Discussion

The pressure-volume relation of the passive inflation phase in figure 4.5 shows that the behavior of the right ventricle is different from that of the left ventricle. During the inflation it is likely that the septum is contributing mainly to the left ventricle mechanics, which might be the reason for the initial decrease of the right ventricle volume. The boundary conditions and
Figure 4.2: The initial model before the passive inflation phase

Figure 4.3: The model after the passive inflation phase
Figure 4.4: The model after the isovolumic contraction phase

Figure 4.5: Cavity volumes and pressures of the left and right ventricle during passive inflation
Figure 4.6: Transmural strains at the end of inflation at five locations around the equator of the ventricles. Note the different scales for the normal and shear strain components.
Figure 4.7: Transmural strains at the end of the isovolumic contraction phase at five locations around the equator of the ventricles. Note the different scales for the normal and shear strain components.
applied pressures of the right ventricle in the current model are determined as a portion of those values in the left ventricle. These conditions need to be investigated further to see if this is the right approach.

During the isovolumic contraction phase, the axial length of the model shortens because the apex is moving towards the basal plane. When looking at the movie [7], this movement seems to be rather jerky. This phenomenon may be a small artifact due to the relatively large pressure steps that were taken. Further experiments need to be done to verify the model predictions with regard to the strains.

The present model does not include the valves, the papillary muscles, a pericardium nor any residual stress, thereby neglecting the loads on the endocardium caused by these aspects. The viscoelasticity of the blood in the capillaries of the myocardium was not taken into account, as were blood mechanics, momentum and gravity.

Finally, the myocardium was considered to be transversely isotropic and no sheet angles were imbedded. The material properties were assumed to be homogeneous throughout the cardiac wall, so no regional variations were taken into account. During the contraction, there was steady state tension and the whole heart was activated at once, while in fact an electrical wave that spirals up from the apex activates the myocardium in 50 ms. These limitations could be solved by coupling the model to a cell model to include the temporal dynamics of the activation sequence and development of contractile tension.
Chapter 5

Discussion and conclusion

5.1 Discussion

In the future a finite element model of the human heart could be used to study different cardiomyopathies, such as myocardial infarction. The mechanical response of the ventricles to an infarct, i.e. a region of fibrosis, could be investigated. A coupled electromechanical model could give some information about the effects of mechanics on the onset and maintenance of ventricular fibrillation.

To make this model more suitable to study the cardiomyopathies described above, the model should be improved and expanded further. First, the fibre distributions used in this study were derived from a study of canine hearts. In the past, canine fibre orientations derived from MRI data have been fitted to a model of the left ventricle [6]. For this model, it is of course desired to use human fibre orientations that were derived from the same heart as the geometrical surface data. In the future, the DT MRI fibre data provided by the National Institute of Health could be fitted to the anatomically realistic finite element model. This would provide a more anatomically accurate description of the human fibres, also these fitted fibre orientations would include a fibre imbrication angle, which is not embedded into the current model. To provide a more accurate representation of the anatomy of the heart, the model can be expanded to include the arteriovenous structures of the basal plane and papillary muscles. To investigate whether the model is suitable for the studies described above, the other phases of the cardiac cycle should be simulated also. Furthermore, the boundary conditions and pressures that were applied on the right ventricle should be investigated further.
5.2 Conclusion

The goal of this project was to develop a geometrical and mathematical model of both ventricles of the human heart. To accurately describe the geometry of these ventricles, the finite element model was based on human DT MRI data. The created model is an accurate representation of the geometry of the human ventricles, but does not include a complete description of the muscle fibres. Two phases of the cardiac cycle were simulated with the model. To make the model suitable for simulating different cardiomyopathies, the model has to be improved and expanded.
Bibliography


Appendix A

Mappings for the DOFs for the elements at the right ventricle insertion

The mappings of the DOFs for node category A, B, C and D, as displayed in figure A.1, are (with v1, v2 and v3 as version 1, 2 and 3):

• For node category A:
  - The nodal coordinates for version 1, 2 and 3 are mapped.
  - Map $\frac{\partial \mathbf{x}}{\partial \xi_1}, v1$ with $\frac{\partial \mathbf{x}}{\partial \xi_1}, v3$, to make the line that crosses node A continuous.
  - Map $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v1$ with $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v3$, to make the surface that crosses node A continuous.
  - Map $\frac{\partial \mathbf{x}}{\partial \xi_2}, v2$ with $\frac{\partial \mathbf{x}}{\partial \xi_3}, v3$, to make the surface across the line continuous.
  - Map $\frac{\partial^2 \mathbf{x}}{\partial \xi_2 \partial \xi_3}, v2$ with $\frac{\partial^2 \mathbf{x}}{\partial \xi_2 \partial \xi_3}, v3$, to make the surface continuous.
  - Map $\frac{\partial \mathbf{x}}{\partial \xi_3}, v1$ with $\frac{\partial \mathbf{x}}{\partial \xi_3}, v2$, to make the surface across the line continuous.
  - Map $\frac{\partial^2 \mathbf{x}}{\partial \xi_3 \partial \xi_2}, v1$ with $\frac{\partial^2 \mathbf{x}}{\partial \xi_3 \partial \xi_2}, v2$, to make the surface continuous.
  - Map the $\frac{\partial \mathbf{x}}{\partial \xi_2}$ of every version, to make the surface across the line continuous.

• For node category B:
  - The nodal coordinates for version 1, 2 and 3 are mapped.
  - Map $\frac{\partial \mathbf{x}}{\partial \xi_1}, v1$ with $\frac{\partial \mathbf{x}}{\partial \xi_1}, v3$, to be of opposite sign, to make the right ventricle less sharp at this node.
Figure A.1: The basal plane of the geometry with mappings for node categories A, B, C and D explained.
- Map $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v_1$ with $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v_3$, to be of opposite sign, to make the right ventricle less sharp at this node.
- Map $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_2$ with $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_3$, to make the surface across the line continuous.
- Map $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v_2$ with $\frac{\partial^2 \mathbf{x}}{\partial \xi_2 \partial \xi_3}, v_3$, to make the surface continuous.
- Map $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_1$ with $\frac{\partial \mathbf{x}}{\partial \xi_2}, v_2$, to make the surface across the line continuous.
- Map $\frac{\partial \mathbf{x}}{\partial \xi_2}$ of every version, to make the surface across the line continuous.

- For node category C:
  - The nodal coordinates for version 1, 2 and 3 are mapped.
  - Map $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_1$ with $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_3$, to be of opposite sign, to make the right ventricle less sharp at this node.
  - Map $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v_1$ with $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v_3$, to be of opposite sign, to make the right ventricle less sharp at this node.
  - Map $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_2$ with $\frac{\partial \mathbf{x}}{\partial \xi_3}, v_3$, to be of opposite sign, to make the surface across the line continuous.
  - Map $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v_2$ with $\frac{\partial^2 \mathbf{x}}{\partial \xi_2 \partial \xi_3}, v_3$, to make the surface continuous.
  - Map $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_1$ with $\frac{\partial \mathbf{x}}{\partial \xi_2}, v_2$, to make the surface across the line continuous.
  - Map the $\frac{\partial \mathbf{x}}{\partial \xi_2}$ of every version, to make the surface across the line continuous.

- For node category D:
  - The nodal coordinates for version 1, 2 and 3 are mapped.
  - Map $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_1$ with $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_3$, to make the line that crosses node D continuous.
  - Map $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v_1$ with $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v_3$, to make the surface across node D continuous.
  - Map $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_2$ with $\frac{\partial \mathbf{x}}{\partial \xi_3}, v_3$, to be of opposite sign, to make the surface across the line continuous.
  - Map $\frac{\partial^2 \mathbf{x}}{\partial \xi_1 \partial \xi_2}, v_2$ with $\frac{\partial^2 \mathbf{x}}{\partial \xi_2 \partial \xi_3}, v_3$, to make the surface continuous.
  - Map $\frac{\partial \mathbf{x}}{\partial \xi_1}, v_1$ with $\frac{\partial \mathbf{x}}{\partial \xi_2}, v_2$, to be of opposite sign, to make the surface across the line continuous.
Figure A.2: The plane of the model, where the right ventricle is closed, that needs special mappings of node B and C (B* and C*).

- Map $\frac{\partial X}{\partial \xi}, v2$ with $\frac{\partial X}{\partial \xi}, v3$, to make the surface across the line continuous.
- Map $\frac{\partial^2 X}{\partial \xi_1 \xi_2}, v2$ with $\frac{\partial^2 X}{\partial \xi_1 \xi_2}, v3$, to make the surface continuous.
- Map the $\frac{\partial X}{\partial \xi_2}$ of every version, to make the surface across the line continuous.

Of the planes that contain the node categories A, B, C and D the plane that is just above the apex has a slightly different shape, as can be seen in figure A.2. This is due to the fact that at this level the septum and the right ventricle free wall come together to close the right ventricle at the bottom. This means that the DOFs of node B* and C* are different from node B and C, i.e. the mapping of $\frac{\partial X}{\partial \xi_1}, v1$ with $\frac{\partial X}{\partial \xi_1}, v3$ and $\frac{\partial^2 X}{\partial \xi_1 \xi_2}, v1$ with $\frac{\partial^2 X}{\partial \xi_1 \xi_2}, v3$ is no longer of opposite sign (as stated above), but of equal sign.