

New Techniques for Visualizing and Evaluating Left Ventricular Performance

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Abstract

Heart diseases remain the biggest killer in the western world and the prognosis after heart failure is poor. Improving the understanding of cardiac mechanics is an essential step towards advancing the diagnosis and treatment of heart diseases. This article explains techniques for visualizing and evaluating the deformation of a left ventricular finite element model. The following contributions are made: we apply techniques traditionally used in solid mechanics and computational fluid dynamics to biomedical data and suggest some improvements and modifications. We obtain new insight into the mechanics of the healthy and diseased left ventricle and we facilitate the understanding of the complex deformation of the heart muscle by novel visualizations.

Key words: Scientific Visualization, Visualization Tools, Strain Tensor Field, Finite Element Model, Cardiac Mechanics

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1 Introduction

One or multiple heart diseases can result in heart failure, which is a clinical syndrome that arises when the heart is unable to pump sufficient blood to meet the metabolic needs of the body at normal filling pressures [1]. The goal of recording and visualizing cardiac data sets is to recognize and predict heart diseases. The cardiac data set used in this work is a finite element model of the human left ventricle developed by Young et al.[2]. Tagged MRI images were used to obtain the myocardial strain which is defined as the pure deformation (without translation and rotation). Abnormalities in the myocardial strain are detectable before first symptoms of a heart attack occur [3] so that measuring and visualizing the strain might represent a useful diagnosis tool. In this paper we compute performance measures and we visualize the myocardial strain field of the left ventricle of a healthy heart and a heart diagnosed with non-ischemic dilated cardiomyopathy. We use a left-ventricular finite element model and a visualization toolkit specifically designed for biomedical models [4]. The visualization techniques novel to this field are explained and the results are discussed and interpreted.

2 Computing Ventricular Performance Measures

The performance of the left ventricle is often specified using various length, surface and volume measures such as its systolic and diastolic volume and its ejection fraction. The volume of a single finite element is obtained by integrating the identity function over the finite element in world coordinates. The calculation is simplified by using the substitution rule of multi-dimensional integration.

$$\int_{\mathbf{x}(\Omega)} d\mathbf{u} = \int_{\Omega} |\det \mathbf{J}(\boldsymbol{\xi})| d\boldsymbol{\xi} \quad (1)$$

where Ω is the unit cube representing the domain of the parent element, $\mathbf{x}(\boldsymbol{\xi})$ is the transformation function from finite element material coordinates to world coordinates and \mathbf{J} is its Jacobian. The resulting integral can be evaluated efficiently using Gaussian Quadrature. Experiments have shown that 3x3x2 gauss points, i.e., 18 evaluations of the Jacobian, give 4-figure accuracy.

One of the most important measures of cardiac performance is the ventricular (blood) volume and the fraction of blood ejected during contraction. In order to apply the volume computation introduced above the left-ventricular cavity is modeled by finite elements which is easily achieved using our toolkit [4]. Table 1 shows that the ventricular volume of

	ED	ES	SV	EF
Healthy heart	87.15	35.08	52.07	59.75%
Sick heart	314.18	277.94	36.23	11.53%

Table 1

Ventricular volume (in cm^3) of the healthy and diseased left ventricle at end-diastole (ED) and end-systole (ES), stroke volume (SV), and ejection fraction (EF).

the healthy heart at end-diastole is about $87cm^3$ and the stroke volume is $52cm^3$ resulting in an ejection fraction of about 60%. These values correspond well with data reported in the medical literature. For the diseased heart a considerable larger end-diastolic volume is observed. However, the stroke volume is only $36.23cm^3$ and about 30% smaller than for the healthy heart. The ejection fraction is only 11.5%. These values indicate a severe impairment of myocardial function.

3 The Visualization of Myocardial Strain

In order to better understand the local deformation of the myocardium more information is required. As a new visualization method for biomedical tensor fields we use hyperstreamlines [5]. The trajectory of a hyperstreamline is a streamline in an eigenvector field which at each point is tangential to the eigenvector at that point. The other two eigenvectors and corresponding eigenvalues of the strain tensor define the axes and lengths of the ellipsoidal cross section of the hyperstreamline. The remaining eigenvalue is colour mapped onto the hyperstreamline.

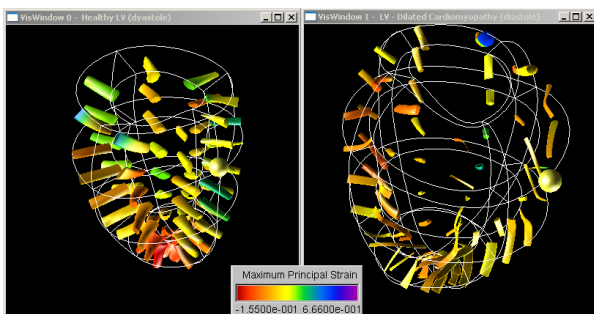


Fig. 1. The strain field in the midwall of the healthy (left) and the diseased (right) left ventricle visualized using hyperstreamlines in the direction of the major principal strain.

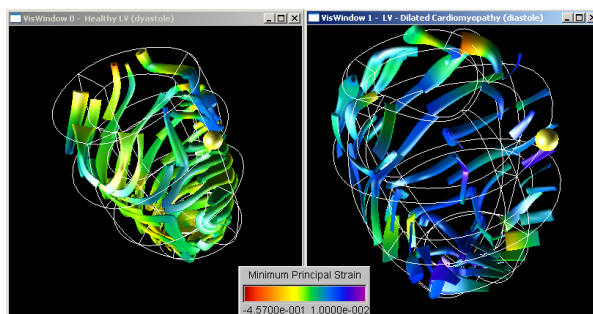


Fig. 2. The strain field in the midwall of the healthy (left) and the diseased (right) left ventricle visualized using hyperstreamlines in the direction of the minor principal strain.

Figure 1 and 2 show hyperstreamlines in the direction of the major and minor principal strain, respectively. The image on the left of figure 1 shows that for the healthy heart the

major principal strain is oriented in radial direction throughout the myocardial wall and that it is positive and increases toward the endocardium. The diameter of the cross section of the hyperstreamline indicates that with the exception of the septal wall (marked by the yellow sphere) the magnitude of the transverse strains increases from the epicardial to the endocardial surface. We are not aware of any previous work showing all these properties with a single image. The minor principal strain of the healthy left ventricle is compressive throughout most of the myocardium and its direction resembles over most of the myocardium a spiral moving toward the apex. This strain direction corresponds well with the rotational motion of the heart described in the medical literature. Note that we have in the inferior-septal region an interesting feature where the hyperstreamlines change suddenly their direction.

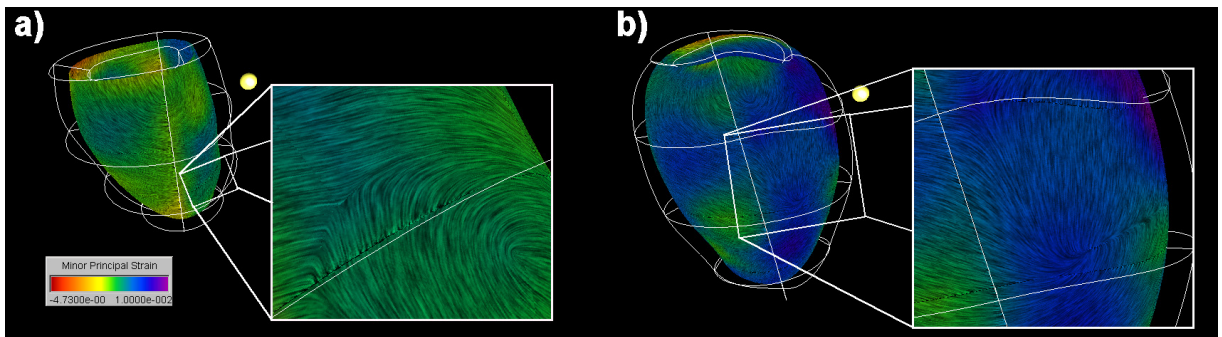


Fig. 3. The minor principal strain (maximum contracting strain) of the healthy (a) and sick (b) heart visualized using Line Integral Convolution.

This feature can be examined in more detail using Line Integral Convolution (LIC) which visualizes a vector field by using curvilinear filters to locally blur an input noise texture along the vector field [6]. We use the direction of the minor principal strain as a vector field and use its magnitude to colour map the texture [4]. Figure 3 (a) shows that the maximum compressive strain in the midmyocardium is predominantly oriented in circumferential direction with a slight downward tilt. Several interesting points exist where the strain suddenly changes direction. Results from tensor analysis show that these points are *degenerate points* for which at least two eigenvalue are equal [7]. An example of such a point is contained within the white rectangle in the figure and is shown enlarged on the right hand side of the image. We found that most of the degenerate points occur on or near the septal wall. The unusual variations in strain orientation might be caused by the right ventricular wall which is connected to the left ventricular wall at both sides of the septum. The strain field of the sick heart contains considerable more degenerate points distributed throughout the myocardium.

We conclude this section with an examination of the distribution of the strains in circumferential and radial direction. These scalar strain values are derived from the strain tensor and quantify the length change of an infinitesimal material volume in the given direction.

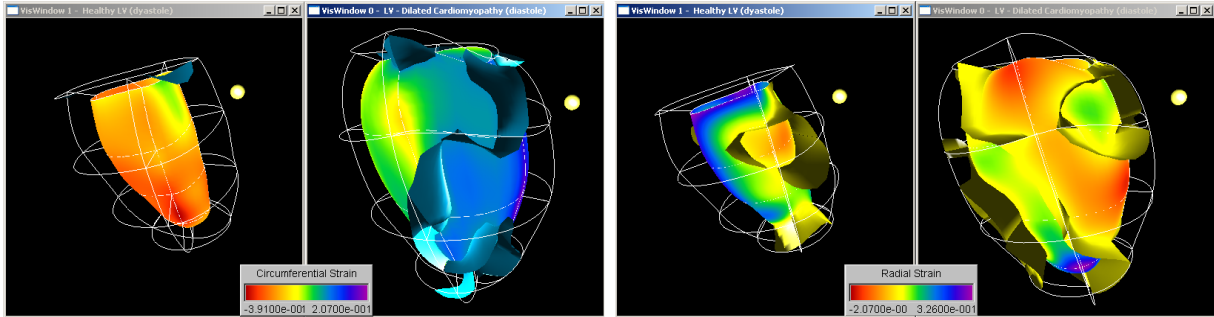


Fig. 4. The normal strain in circumferential (left) and radial (right) direction on the endocardial surface of the healthy and sick heart.

Negative strain values are interpreted as a local shortening of the myocardium and positive strain values as a local elongation. Figure 4 visualizes the circumferential (left) and radial (right) strains on the endocardial surface using colour mapping and shows additionally the 0-isosurface, which separates contracting and expanding regions. The isosurface was computed with a modified *Marching Cubes* algorithm in material space [8]. The images show clearly that the healthy left ventricle contracts in circumferential and expands in radial direction except for some parts of the model boundary and, for the radial strain, three small cylindrical regions at the apex and the septal and lateral wall. For the diseased heart the lateral wall and part of the anterior and inferior wall contract in circumferential direction. Wall thickening is observed in the basal-lateral wall, the basal-septal wall and in parts of the anterior and inferior wall. The rest of the myocardium shows an abnormal deformation. As a result of the strain distribution the ventricle does not contract evenly but rather performs a shape change.

4 Conclusion

Visualizing the strain field improves the understanding of the complex deformation of the heart muscle. Using techniques new to the biomedical field offers additional insight. The visual information can be supplemented by computing ventricular performance measures which are easily obtained from the finite element model using numerical integration. The visualization of the healthy heart confirms observations previously reported in the literature. Using hyperstreamlines makes it possible to visualize complex deformation behaviour in a single image. Line integral convolution uncovers the presence of degenerate points at which the principal strains suddenly change direction. Further investigations are necessary to find the relationship between degenerate points, fiber structure, and the ventricular anatomy. Furthermore we want to explore their significance (if any) for diagnosing heart diseases. Visualizing a ventricle with dilated cardiomyopathy showed that

the deformation of the lateral wall resembles most closely the expected motion whereas the septal wall behaved almost contrary to the expected deformation. Very large negative shear strains were recorded in the anterior-basal wall of the ventricle. The combined effect of these deformations seems to be a pumping action by shape deformation (from circular to ellipsoidal cross section) rather than by contraction.

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